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ARIZONA MEDICINE
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Original ARTICLES

SOME RECENT DEVELOPMENTS IN RABIES CONTROL

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Tucson, Arizona

RABIES in man is an invariably fatal disease, but fortunately the number of individuals dying of the disease is quite small. In the United States of America in the period between 1949 and 1954, the annual reported deaths from rabies have varied from 10 to 24 per year. These figures give little account, however, of the heavy toll exacted by rabies in the form of large sums expended for (1) human immunizations, (2) cost of services to investigate animal bites, (3) quarantine measures and stray animal collections, (4) laboratory and administrative services, and (5) loss of livestock and valuable pets. This takes no account of the anxiety and suffering of the thousands of persons and their families who annually undergo prolonged courses of vaccine treatment due to exposure to rabies infection. When all these considerations are taken into account, rabies must certainly be included among the major problems of those concerned with protecting the health of human and animal populations.

EPIDEMIOLOGY

There are two epidemiological types of rabies – the natural disease as it occurs in wild animals and the urban type which is maintained in domestic dogs.(1) The virus is often present in the saliva of rabid animals and consequently is most commonly transmitted by a bite. Under favorable conditions the virus when introduced into a wound becomes established in nerve tissue and migrates to the brain where, after an incubation period of from ten days to several months, it produces an acute, highly fatal encephalitis. The probability of human infection is dependent upon the concentration of the virus in the saliva of the biting animal; the site of the bite on the body (bites on the head, neck. and face are the most dangerous; the hands, feet, arms, and legs are next in importance; and on the trunk least dangerous); the depth of the bite; the multiplicity of the bite; and the possible interposition of clothing.

Rabies is endemic throughout most of the U.S.A. The intensity of infection varies from area to area depending on the density of wild-life populations and on the efficiency with which control programs are maintained. The situation is well illustrated by the outbreak of an epizootic of rabies that began in New York State in 1943.(2) In Table I, the annual reported incidence of rabies in New York State from 1943-1947 is given. The disease increased during this period markedly in foxes and in cattle, but decreased sharply in dogs due to mass immunization campaigns. The infection was still being frequently encountered among foxes and cattle after rabies in dogs had subsided.

Some idea of the extent of the rabies problem in Texas can be gained from a study of the data summarized in Table II.(3) With the extensive prevalence of rabies as indicated by these figures, it is not surprising to learn that in 1953, 2,675 fourteen-injection human rabies treatments were supplied to physicians for administration in Texas.

Recently in California, skunks have been commonly affected with rabies infection among the wild animals, while dogs were by far most frequently attacked by rabies among the domestic animals. Thus of a total of 425 reported cases of animal rabies in California during 1955,

246 were among dogs and 141 among skunks.(4)

In Arizona, the last human case of rabies reported to the Health Department was that of a three year old child from Yuma. This case occurred three years ago.(5) The incidence of rabies among animals in this state appears to be low according to the data given in Table III.(6) Most cases of rabies in wild animals in Arizona have occurred in the low-altitude areas of the southern counties and have been discovered among coyotes, skunks, foxes, and bobcats. Cattle, too, have been frequently affected. Since animals are seldom submitted for examination in the laboratory unless they have approached residences, usually during the day, and have attacked domestic animals or humans, it seems obvious that the actual level of rabies infection among wild animals is much higher than is reported.

In recent years, interest has centered around the possible role of bats in the epidemiology of rabies. In the Americas, there are three general groupings of bats. Based upon feeding habits, they are: the carnivorous or blood-lapping vampire (**Desmodus sp.**); the fruit eating (**Artibeus sp.**); and the insectivorous. The insectivorous bats are further grouped as colony bats (**Tadarida sp.**) which congregate and live in caves, attics, and belfries; and the free-living bats (**Lasiurus sp.**) which live singly in trees and bushes. In the New World, the vampire and fruit-eating bats are confined to Latin America.

It has long been known that rabies is common among and transmitted by the bite of vampire bats. In 1908, ranchers in southern Brazil observed that bats were attacking and biting livestock in the daytime and that animals thus bitten soon died of a paralytic disease. Some years later, some of the cattle affected with this disease were proved to be infected with rabies virus and shortly thereafter rabies virus was isolated from vampire bats in that area. In 1925, a similar situation was reported in Trinidad, B. W. I. Several thousand cattle died and 55 fatal human cases occurred. All of these were thought to have been infected by the bites of vampire bats. In Mexico, outbreaks of a paralytic disease in cattle have been observed in the west central states for more than 40 years. During a field study in the state of Michoacan in 1944, rabies virus was isolated from a paralyzed cow and from the salivary glands of vampire bats captured in a cave nearby.(1)

Rabies in vampire bats may manifest itself in the classical furious or paralytic type common to all mammals. These bats may recover, but more usually they die. Some bats after infection develop a carrier state without exhibiting any evident illness. How long the naturally infected bat may live and act as a carrier is not known, but it appears probable that it may be for many months.(7) Living together as they do in large colonies in caves, there is abundant opportunity for the maintenance of the infection in the colony for long periods of time and for passing the virus to other bats.

Vampire bats have a wide range extending from Argentina to Central America and Mexico. There is good reason to believe that their distribution now encompasses a far greater area than it once did, favored by an increase in the cattle population. Thus far, the nearest locality to the U. S. border where vampire bats have been found is Guadalupe Cave near Linares, Nuevo Leon, only 100 miles from the frontier.(7)

Among bats, rabies is not limited to vampires. At least seven other species have been found infected in nature. The first positive report of bat rabies in this country came from Florida in 1953 when Negri bodies were found in the brain of a Florida yellow bat which had attacked a child. Later, rabies virus was isolated from the brains of five additional Florida yellow bats and from one Seminole bat. These two species are non-colonial, insectivorous bats, indigenous to the southeastern United States.(8)

A few weeks after the Florida report, rabies virus was recovered from the brain of a bat, identified as probably the red hoary bat (**Lasiurus cinereus**), which had attacked a woman in Pennsylvania. This bat is of the migrating type, going probably to central Mexico for the winter months.(9)

In 1954, rabies virus was obtained from the brain of a sick bat found in a flower bed in Montana. This bat was identified as belonging to a large insectivorous species common to western U. S. A. (**Eptesicus fuscus pallidus Young**). It is not known to migrate, but hibernates in caves.(10)

Strenuous efforts have recently been made to study the relationship of bats to the rabies problem in several states. In Texas, interest has been focussed on the Mexican free-tailed

bat (**Tadarida mexicana**). The caves of central Texas harbor millions of these bats living in huge colonies, many of which migrate to Mexico in the cold season. Consequently exchange of infection between these insectivores and the vampires is an ever-present possibility. During a two-year study, some 2,000 bats have been tested for rabies infection in Texas. Bats of ten different species were among those tested. Rabies virus was repeatedly found in the brains and salivary glands of the Mexican free-tailed bat. The only other species found infected was a single specimen of the "red bat" (**Lasiurus borealis**). (11)

In the caves harboring bat colonies, evidence was found that at certain seasons raccoons and other predatory animals feed on bats, especially on those which have fallen to the cave floor. This intimate contact between carnivorous animals and infected bats provides a hypothetical mechanism for the passage of rabies infection in nature.(12)

Another study of bat rabies has been carried out recently by officers stationed at Brooke Medical Center, Fort Sam Houston, Texas. A total of 1,247 bats representing nine species were collected from Texas, Arkansas, Louisiana, New Mexico, and from several points in Old Mexico. Virus was discovered in the brains of the free-living species (**Lasiurus borealis borealis** and **Antrozous pallidus pallidus**) in Texas, as well as from **Tadarida brasiliensis mexicana** and **Myotis velifer incautus** in New Mexico and Texas, and from **Tadarida brasiliensis cynocephala** in Louisiana.(13)

In California in the summer of 1956, two instances of bat rabies have been reported. One was in a hoary bat (**Lasiurus cinereus**) killed while attacking a dog in Shasta County, and the other a bat of the genus **Myotis,** species unitentified, killed when attacking a boy in Santa Clara County. Both bats were proved to be rabid by laboratory examination.(4)

It would appear from these studies that rabies is widely prevalent among bats in this country, but a great deal of investigation remains to be done before the epidemiological role of bats in rabies can be properly assessed. Since it is clear that bats may harbor rabies virus and yet show no signs of illness or abnormality of habit, any bite inflicted by a bat, molested or otherwise, should be considered as possible exposure to rabies virus.

Even though only fragmentary information is available, it would appear that bat colonies may serve as the true reservoirs of rabies virus. From these reservoirs, infection may spill over periodically into wild animals, either infected by the bites of rabid bats or from the eating of bats carrying virus. Once introduced among coyotes, foxes, and other carnivores, rabies sweeps through the animals in the bush, the intensity of the epizootic depending on the density of the animal population. Livestock and pets on ranches, in farm yards, and in towns may be infected by contact with rabid wild animals. Domestic dogs revert easily to the semiwild state and stray dogs increase rapidly in any urban community unless there is an organized effort to destroy them. The propagation of rabies in dogs is dependent to a large extent on the presence of many stray dogs in urban communities.

CONTROL MEASURES

At the present time, control of rabies among wildlife in situations where the disease exists at low levels or on an endemic basis seems impracticable. When sharp outbreaks or epizootics occur, well-organized campaigns for the reduction of excessive numbers of wildlife vectors should be undertaken. Such outbreaks are usually associated with high densities of vector populations, and the objective should be to bring the number of animal vectors down to a level that would no longer support the rapid spread of the disease. These programs should be directed by experienced men, expert in the techniques of trapping, shooting, gassing of dens, and in the use of poisons.

Control programs should be set up on a statewide basis and coordination of activities is essential for success. Whenever possible, a well-trained veterinary officer should be given responsibility for the administration of the state program.

The attack against an infectious disease like rabies must necessarily depend upon adequate facilities for rapid and accurate diagnostic procedures. Personnel experienced in the techniques for demonstrating Negri bodies in brain specimens and in virus isolation by animal inoculation should be available in State Public Health Laboratories. The importance of animal inoculation tests for the isolation of virus from suspected brain tissue in Negri-negative specimens cannot be overemphasized. Extensive studies

have shown that in large series of routine specimens submitted for diagnosis that 10-15 per cent of these cases proved positive by mouse inoculation had been missed by direct microscopic examination for Negri bodies.

Local rabies control functions best on a county-wide basis. These programs must include: (1) Mass immunization of dogs with the objective of vaccinating at least 70 per cent of the dogs in every town. (2) Elimination of all stray dogs. These ownerless animals should be collected and held for a few days at the local pound or animal shelter. If unclaimed, the strays should be destroyed in a humane manner. (3) A licensing system for all dogs; if properly enforced, this system aids in the identification of ownerless dogs and helps to defray the expenses of the control campaign. (4) Quarantine of animals known to have been bitten by rabid or suspicious beasts; since the incubation of rabies is often prolonged, these animals must be kept under observation for at least three months.

Employment of the measures outlined above has repeatedly given rapid and satisfactory reduction in the incidence of rabies among dogs in affected communities. Even by the use of inactivated nerve-tissue vaccine, which confers immunity to dogs for a period of about one year, good results were obtained. Now that the living chick-embryo vaccine (Flury strain) is available, control results are even more satisfactory. This vaccine gives good protection to dogs for at least three years. All dogs three months of age and older can be effectively immunized by a single inoculation of this product. Many reports of the successful reduction of rabies among the dog population of American communities have been published. Recently, the results of two dramatic demonstrations of the efficacy of this control method on a national basis in Malaya and in Israel have become available.(14), (15)

A most important aspect of any successful rabies control program must be the education of the public. Unfortunately, people are inclined to be indifferent, or all too often in the case of pet owners, to be belligerent toward a rational plan to immunize the dog population. It is only through an understanding of the whole problem that people can be persuaded to cooperate with the health authorities on a continuing basis to keep rabies down to a minimum level in the community. Probably more can be done through the efforts of veterinarians than by any appeal based primarily on the human aspects of the problem. Of course, the public should also be warned about avoiding wild animals that appear ill or whose behavior is abnormal.

PREVENTION OF HUMAN RABIES

Infection from the dog or domestic cat accounts for all but 1-2 per cent of human infections. The first line of defense, therefore, is an effective campaign to render such pets immune to rabies by vaccination.

There are no satisfactory data available concerning the attack rate for rabies in man following exposure by the bite of a rabid dog. Since Pasteur first introduced his treatment in 1885, it has been the practice to give such anti-rabic vaccine to all persons exposed whenever possible. An indication of the overall attack rate in the untreated individual may be obtained from the data of Schuder who reported 1,325 deaths from rabies in 14,959 persons bitten by rabid animals (9 per cent) before vaccination therapy was available. Other reporters present a less optimistic picture. In southern India from 1946-1954, among 213 persons bitten by animals whose infectivity was proved by death from rabies of one or more of the persons bitten and who received a complete course of treatment with Semple vaccine 16 or 7.5 per cent died of rabies. At the same time, of 85 persons who remained untreated after a similar exposure, 36 or 42.3 per cent succumbed to rabies.(16) It would appear that man is relatively susceptible to rabies and under some conditions the attack rate is very high.

As for the diagnosis of the disease in man, one should bear in mind that some patients do not show the classical clinical symptoms. In such individuals, rabies is likely to be mistaken for fulminating poliomyelitis. The diagnosis in these cases can only be made by the laboratory after death or by the inoculation of saliva into the brains of laboratory animals.

In order to prevent the development of rabies in man following exposure, it is necessary to achieve the equivalent of intracerebral resistance. The immunity produced by a series of vaccine injections must be sufficient to stop the multiplication of the virus in the nervous system.(17) It requires 3-4 weeks to develop maximum immunity after beginning the post-ex-

posure vaccine treatment, as determined by the rate of development of virus-neutralizing antibodies in the blood. In cases of severe bites, especially around the head, the incubation period in man may be short, sometimes less than two weeks, so the vaccine treatment may not prevent the development of rabies. Most of the failures of vaccine to protect come in this category.

Since Pasteur's original vaccine prepared from infected spinal cords of rabbits dried in jars containing potash, many other types of vaccine, containing both active virus and virus killed by various methods, have been introduced. It was not until the white mouse became available as an experimental animal that the immunizing potency of rabies vaccines could be accurately tested. Now all rabies vaccines marketed in this country must be tested for immunizing potency according to a method developed by the U. S. Public Health Service.(18) The vaccine most commonly used now in the U. S. A. is the Semple type. It is packaged in 14 doses of 2 cc. each of a 4 or 5 per cent rabbit brain suspension in saline solution containing 0.25 per cent phenol. The usual treatment consists of 14 daily injections of 2 cc. of vaccine, given subcutaneously in the abdominal wall.

In addition to occasional failures of the vaccine to protect on account of an unusually short period of incubation of the disease, there is danger of sensitizing to rabbit-brain tissue which sometimes produces serious allergic reactions. Paralytic phenomena, as often as 1 in 600 in some reported series, may follow the administration of rabies vaccine. These reactions include peripheral neuritis, dorso-lumbar myelitis, and paralysis of the Landry's type. Some of these paralytic cases are fatal, but more often they recover either fully or with some residual disability.

In a recent study, Koprowski and LeBell(19) demonstrated complement-fixing antibodies against rabbit-brain tissue in 50 per cent of the sera of a group of persons who had received a course of 14 inoculations of Semple-type vaccine. Those receiving only seven inoculations developed little or no such antibody. In three individuals with post-vaccinal neurological complications, the anti-rabbit brain antibody titres were particularly high.

With these two disadvantages of conventional anti-rabies vaccines in mind, i.e., failure to produce immunity rapidly and the danger of post-vaccination reactions, work has gone forward on studying the possibility of using prophylactic immune serum in the treatment of persons exposed to rabies. A concentrated hyperimmune serum produced in horses is now available commercially. Experimental work has definitely shown the superiority of this hyperimmune serum, especially when combined with a course of vaccine inoculations, over vaccine alone in the treatment of animals infected with rabies virus.(20) As the antibodies in the serum provide immediate passive immunity, use of the serum is recommended in severe bites, especially around the head, or in persons bitten badly by wild animals. It seems probable, too, that with the use of the serum, the number of doses of vaccine can be reduced to perhaps seven. Experience in this direction is being obtained in several states.

Since the anti-rabies serum is prepared in horses, it is necessary to test each patient for sensitivity before the serum is administered. If sensitivity is encountered and the need for using serum is imperative, the usual measures to desensitize the patient must be carried out. The recommended dose of serum is 0.5 cc. per kilogram of body weight. In cases of very severe exposure, larger doses of serum are indicated. In a series of 32 patients who received hyperimmune serum in Alabama, eight developed serum sickness of varying intensity with the reaction being severe in three of them.(21)

Another attempt to improve the rabies vaccine has been in the direction of removing animal brain tissue by cultivating the vaccine virus in duck embryos. A fixed strain of rabies virus is injected into developing duck embryos to secure multiplication of the virus. After a few days of incubation to provide for maximum growth, the virus is harvested and the vaccine prepared from the duck-embryo tissue.(22) So far, experience with this type of vaccine is not sufficiently advanced to permit judgment.

Perhaps the most significant advance in rabies research for many years has been the development of a living virus vaccine prepared from chick embryos, i.e., the Flury strain. This strain was isolated from the spinal cord of a young girl who died in Georgia after exposure to saliva of a rabid dog.(23) After prolonged passage from brain to brain in one-day old chicks, the virus strain was transferred to developing chick

embryos. Koprowski and Black(24) in 1950 found that virus material from the 40th-50th chick-embryo passage had become non-pathogenic for dogs when inoculated intramuscularly. A single injection of an appropriate dose of the chick-embryo virus resulted in good protection in dogs against subsequent inoculation of virulent strains of street rabies virus. The obvious advantages of this method of immunizing dogs were quickly perceived and the avianized Flury strain vaccine has been in general use for several years. As has already been pointed out above, the results of mass vaccination campaigns for dogs have been eminently satisfactory, and it is now known that immunity produced by this method endures for at least three years.

In the course of prolonged cultivation of the Flury strain in chick embryos, it was found that between the 172nd-174th passages the virus lost its capacity to produce fatal infection in adult mice, dogs, and rabbits even upon intracerebral inoculation.(25) This high-passage material was still antigenic in dogs and cattle upon the inoculation of a single dose intramuscularly. This virus material inoculated into the brain proved pathogenic for young mice 3-8 days of age, so there was still available a test animal for demonstrating the quantity of active virus present in any lot of this greatly modified vaccine.

The possibility of employing this high-passage Flury strain for human immunization soon came under consideration. After some preliminary experiments by Koprowski and his associates at the Lederle Laboratories, a group of research workers at Tulane University undertook to study the effects of the new vaccine in human volunteers.(26) Ninety-eight persons received from 1-10 intramuscular doses of chick-embryo virus containing material of between the 179th-181st passages. Each dose consisted of 2 grams of tissue suspension. No reactions of any consequence resulted from the vaccinations. Tests done on sera taken at intervals after inoculation of the vaccine showed that specific antibodies could be consistently demonstrated if vaccine had been given in sufficient quantities. Three doses of vaccine were usually sufficient to produce a good antibody level. Since such large quantities of the virus-containing tissue is required to stimulate immune response, it appears probable that there is no multiplication of the virus following parenteral inoculation into the human.

Trials of the high egg-passage Flury strain for human immunization are continuing. Experience so far indicates that this method will be safe and useful. Some years will probably be required to collect enough evidence to provide an adequate basis to assess the proper place of this vaccine in the prevention of human rabies.

POST-EXPOSURE TREATMENT OF MAN

The Expert Committee on Rabies of the World Health Organization in its 1954 report(27) strongly recommends the immediate treatment of all bite wounds inflicted by animals, especially those suspected of being rabid, by thorough cleansing with soap or detergent solution. Such treatment does not preclude the subsequent use

TABLE I*

Reported Annual Incidence Of Rabies In New York State, Exclusive Of New York City, 1943-1947, By Species

Year	Dogs	Cattle	Foxes	Human	Other	Total
1943	176	10	1	0	2	189
1944	233	47	15	1	18	314
1945	503	87	50	1	22	663
1946	377	440	308	0	49	1,174
1947**	40	167	218	0	31	456

*Taken from: Korns and Zeissig, Amer. J. Pub. Health, 1948, 38, 50-65.

**Through September 24.

TABLE II*

Reported Annual Incidence Of Rabies In Texas, 1948-1953,** By Species

Year	Dogs	Cattle	Foxes	Skunks
1944	863	24	3	1
1945	763	17	0	1
1946	1,004	29	16	15
1947	956	25	40	7
1948	1,203	48	75	73
1949	712	43	129	44
1950	863	61	160	43
1951	970	61	181	58
1952	1,009	51	161	74
1953	1,029	53	150	49

*Taken from: Irons, Eads, Sullivan, and Grimes, Texas Reports on Biology and Medicine, 1954, 12, 489-499.

**During this 10-year period, there were 29 human deaths from rabies in Texas.

TABLE III*

Rabies Positive Examinations Among Animals In Arizona, 1946-1955

Year	Dogs	Other Animals	Total
1946	11	10	21
1947	16	8	24
1948	20	3	23
1949	24	4	28
1950	22	2	24
1951	10	1	11
1952	7	4	11
1953	26	6	32
1954	24	6	30
1955	8	12	20
Total	168	56	224

*Examinations carried out by Laboratories of Arizona State Department of Health, Phoenix.

of strong mineral acids, such as nitric acid, which may be introduced into the depths of puncture wounds that cannot be reached satisfactorily with soap or detergents.

Because of the remarkably low mortality rate among treated persons, the committee recommends the use of vaccines for the prevention of rabies. Vaccine treatment should not be given, however, unless there is good evidence of exposure to rabies. As a guide to physicians, the W. H. O. Committee has prepared a statement summarizing the indications for specific post-exposure treatment.(27) In Table IV, these recommendations are reproduced.

SUMMARY

Although rabies now occupies a relatively unimportant place among human diseases, it continues to be a major source of anxiety, and its presence in the country on an endemic basis results in large expenditures of money for control programs.

In recent years, rabies infection in bats has been found to be widely prevalent in many parts of the country. It appears that bats in colonies may be the true reservoir of the disease, as these animals sometimes harbor the virus for months without prejudice to their health.

Rabies occurs also among wild carnivores, such as coyotes, foxes, and bobcats, and presumably spreads from these animals to domestic pets.

TABLE IV*
Indications For Specific Post-Exposure Treatment

Nature of exposure	Condition of biting animal		Recommended treatment
	At time of exposure	During observation period of 10 days	
I. No lesions: indirect contact only	Rabid	–	None**
II. Licks: (1) unabraded skin	Rabid	–	None**
(2) abraded skin and abraded or unabraded mucosa	(a) Healthy	Healthy	None
	(b) Healthy	Clinical signs of rabies or proved rabid	Start vaccine at first signs of rabies in animal
	(c) Signs suggestive of rabies	Healthy	Start vaccine immediately; stop treatment if animal is normal on 5th day after exposure***
	(d) Rabid, escaped, killed or unknown	–	Start vaccine immediately
III. Bites: (1) Simple exposure	(a) Healthy	Healthy	None
	(b) Healthy	Clinical signs of rabies or proved rabid	Start vaccine at first signs of rabies in animal
	(c) Signs suggestive of rabies	Healthy	Start vaccine immediately; stop treatment if animal is normal on 5th day after exposure***
	(d) Rabid, escaped, killed, or unknown; or any bite by wolf, jackal, fox, or other wild animal	–	Start vaccine immediately
(2) Severe exposure; (multiple; or face, head, or neck bites)	(a) Healthy	Healthy	Hyperimmune serum immediately; no vaccine as long as animal remains normal.
	(b) Healthy	Clinical signs of rabies or proved rabid	Hyperimmune serum immediately; start vaccine at first sign of rabies.
	(c) Signs suggestive of rabies	Healthy	Hyperimmune serum immediately, followed by vaccine; vaccine may be stopped if animal is normal on 5th day after exposure.
	(d) Rabid, escaped, killed, or unknown. Any bite by wild animal	–	Hyperimmune serum immediately, followed by vaccine

*Taken from: W. H. O. Techn. Rep. Ser., No. 82, 1954, 12.
**Start vaccine immediately in young children and in patients where a reliable history cannot be obtained.
***An alternative treatment would be to give hyperimmune serum and not start vaccine as long as the animal remained normal.
Note: To be effective hyperimmune serum must be given within 72 hours of exposure. Dose: 0.5 ml per kg of body-weight.
These indications apply equally well whether or not the biting animal has been previously vaccinated.

The vast majority of human cases occur from exposure to the bites of dogs and cats. The most effective method of preventing such cases is through concerted state- and county-wide campaigns to immunize dogs against rabies. Fortunately, a single dose of avianized vaccine produces a high degree of immunity in dogs that persists for at least three years.

In man, anti-rabic vaccine given after exposure sometimes fails to prevent rabies when the incubation period is short. These failures usually occur in persons with severe animal bites around the head and face. The use of hyper-immune serum to establish passive immunity soon after such exposures has been found to be effective.

Neurological complications are occasionally noted following the use of nerve-tissue rabies vaccines. To avoid the risk of such accidents, the use of immune serum with a reduced number of doses of vaccine is being advocated. Also research is going forward to produce chick-embryo vaccines for human use. Preliminary results with such an avianized vaccine are most encouraging.

Rabies in man is a preventable disease. Its control depends to a large extent on an educational campaign to acquaint the public with the essential aspects of the problem.

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THE LOCALIZATION OF OBSCURE GASTROINTESTINAL BLEEDING*

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THE PROBLEM of the management of obscure gastrointestinal bleeding is one that is constantly with the general practitioner, the internist, and the surgeon. Much has been written about it and much will continue to be written, because insofar as one can foresee it will always remain a problem and there will be no easy answer found either from the standpoint of localization or treatment.

The recent trend toward increased medical care confronts the internist with the responsibility for many patients with low grade hemorrhage from the gastrointestinal tract which previously would have been undetected. Similarly because of the increased availability of whole blood, many patients with recurring severe gastrointestinal bleeding who previously would have succumbed early in their illness, are kept alive as diagnostic problems. These trends present us with the increasing problem and responsibility of keeping the quality of our management of gastrointestinal bleeding abreast of the advances which have been made in other medical fields.

The purpose of this paper is to discuss methods of diagnosis which can be profitably used and to emphasize the importance of each physician developing a plan of management for cases of gastrointestinal bleeding which is positive in character. The answer to this over-all problem certainly does not lie in handing the X-ray man the problem of making the diagnosis or of passing it on to the surgeon when this does not produce the desired results.

At the present time diagnostic difficulties usually fall into two categories: (1) those cases in which the x-ray studies show no evident cause for the bleeding, and (2) those cases in which the x-ray studies show one or more possible causes and where the question exists if either or any of the possible causes is actually responsible. This latter situation frequently occurs in older age groups and one may literally have four or five possible sources of the bleeding made evident by x-ray examination.

In trying to arrive at a diagnosis it is of great help to determine the level at which the bleeding is occurring. We approach this in many ways. From the standpoint of history is there anything to indicate a peptic ulcer? Is there a history of alcoholism? Is there a history of bleeding from other organs or in other members of the family which would suggest familial hereditary teliangectasis? Is there a history of hemophilia? Such facts may give suggestive and helpful information. In the physical examination one may find purpura, petechiae, or other evidences of blood dyscrasia. Abdominal scars may suggest previous surgical procedures for peptic ulcer. The stigmata of cirrhosis may be present and suggest the possibility of varices as a source of the bleeding. Careful examination of the nose, throat, teeth, and the rectosigmoid are often rewarding. The laboratory studies in addition to giving information relative to the amount of blood lost may also reveal evidences of blood dyscrasia, cirrhosis, uremia, or of syphilis.

All of the above information either in a positive or a negative sense is of value in arriving at a correct diagnosis. In most cases this information plus that derived by x-ray study will give a firm answer or a highly educated guess.

In those cases in which a satisfactory answer cannot be obtained from this approach, further localizing studies will be required. The type of such studies which will be most suitable in the individual case will depend upon whether the bleeding is coming from above the duodenal bulb, between the doudenal bulb and the ileocecal valve, or between the ileocecal valve and the rectosigmoid. If hematemesis is present the bleeding point is normally above the ligament of Treitz. However, there are frequent cases of bleeding from the esophagus, stomach, and duodenum in whom there is no hematemesis. The question "Do you vomit easily?" often gives some information in the evaluation of the sig-

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nificance of absence of hematemesis. The person who vomits easily will usually do so if a significant amount of blood is introduced into the stomach. A useful procedure in patients who are bleeding and who are not vomiting is to insert a tube into the stomach to observe the character of its contents. A positive finding of blood indicates bleeding superior to the jejunum. A negative finding, however, does not necessarily exclude bleeding below the stomach. A case of massive bleeding from a duodenal ulcer was recently seen in whom the gastric contents were entirely free of blood during the period of active bleeding. Careful examination of the character of the blood in the stool is of some value. While it is not possible to distinguish in this way between bleeding from the upper gastrointestinal tract and the proximal colon, blood that is streaked through a formed stool often indicates a source distal to the cecum. Blood on the surface of the stool only usually means bleeding from the rectum or sigmoid. Bleeding that accompanies a formed stool and continues after the completion of evacuation is usually anal in origin.

If one suspects on the basis of history, x-ray findings, hematemesis, or the proved presence of blood in the stomach, that the source of the bleeding is above the jejunum, further localization may be obtained in several ways. Although it antedates the use of x-ray and is largely a forgotten procedure, the string test of Einhorn is a simple and often valuable procedure in such cases. When positive it distinguishes very effectively between bleeding from the esophagus, upper stomach, antrum, and duodenal bulb. When negative it is of no localizing value, but if properly done is useful in that it reliably demonstrates the cessation of bleeding above the ampulla of Vater. Localization if obtained in this way can be helpful in guiding both the x-ray man and the endoscopist to a particular area for more careful study.

The string test, as modified by Rappaport, consists of the use of a piece of 4 ply wool yarn 33 inches in length, weighted at the end with a size 7 split shot and with radio-opaque markers at measured intervals. It is knotted 30 inches from the weighted end, moistened, and swallowed. The knot is taped to the corner of the mouth. This is done in the evening and the string removed the following morning before breakfast. An x-ray taken prior to removal will confirm the position of the yarn. In the presence of very active bleeding this test is valueless except to show that such bleeding is still in progress, since the entire distal portion of the yarn will be soaked with blood and no localization can be obtained. If the yarn when withdrawn shows no evidence of blood staining, the test again has no value except to show that bleeding has stopped which may not be evident clinically at the time. A positive test depends upon a portion of the yarn coming in contact with a lesion which will bleed sufficiently on such contact to stain the adjacent segment of the yarn. Bile staining of the distal segment will indicate that the yarn has been in proper position with the tip in the duodenum. In this circumstance a blood stained segment at 16 inches or less will indicate an esophageal lesion; at 21 to 23 inches, an antral lesion; and at 24 to 25 inches, the duodenal bulb. Since the distance to the pylorus varies considerably an x-ray confirmation of position is desirable for lesions in the distal stomach or proximal duodenum. This test is normally quite simple to perform and is well tolerated by the average patient. It can be done nightly if necessary until a satisfactory answer is obtained or until all bleeding has stopped.

Finally the direct approach which is being taken with increasing frequency in cases of upper abdominal bleeding is combined endoscopy, i.e. esophagoscopy followed by gastroscopy, at the same sitting. This is being done more and more commonly at the time of the bleeding at which the information is urgently desired, and because certain sources of bleeding, such as from superficial ulceration or mucosal oozing in hypertrophic gastritis, are evanescent in their presence.

The need for such studies in cases where other studies are non-revealing is obvious. They are often equally helpful in cases where multiple diagnostic possibilities do exist. For example, in the presence of cirrhosis possibly no more than 40% of cases with bleeding manifested by hematemesis actually bleed from varices. When such bleeding does occur from varices it is usually from the lower esophagus, but may be rather high in the esophagus and not uncommonly comes from the varicosities in the cardia of the stomach. The remaining 60% of people with cirrhosis who bleed are about evenly divided between those that bleed from peptic ulcer and those that bleed from hypertrophic

gastritis. The speaker recently saw a case of cirrhosis of the liver, confirmed at surgery, with massive hematemesis in whom two x-ray studies had proved negative. Esophagoscopy was done and no varices or other source of esophageal bleeding was found. Gastroscopy was then done and a 1 cm. shallow ulcer filled with clotted blood was observed on the poserior wall of the body of the stomach. The patient was again x-rayed with negative findings. At surgery a thrombosed gaping vessel was found in the base of the ulcer. A second type of case in which endoscopy is of value is the patient with hematemesis who has x-ray evidence of a multiplicity of lesions. A common example is a combination of hiatus hernia, peptic ulcer, with or without either a gastric or duodenal diverticulum. Only by direct localization of the bleeding to one or the other of the lesions can a correct diagnosis be made. Unfortunately, endoscopy cannot reach duodenal lesions, but here again the string test of Einhorn, if positive, is useful in demonstrating a bleeding lesion in the duodenal bulb. If a bleeding lesion distal to the bulb and in the small intestine is suspected, intubation may prove a valuable procedure. The speaker prefers for this purpose a single lumened plastic tube twelve feet in length and with a mercury filled bag fastened to the tip. This is inserted into the stomach and allowed to pass to the distal ileum. Specimens are taken for gross examination and chemical test from the stomach and each foot distal thereto, where it is possible to obtain a specimen. If gross blood is encountered, or if there is a significant increase in the amount of occult blood obtained, an x-ray is taken to further localize the tip. Barium may be injected if desired. The stools are studied for gross and occult blood daily for a period of three days before and if the study is negative for three days after the test. Similar information can be obtained with less pitfalls, but also with less patient acceptance, from a four lumened tube inserted to the terminal ileum and so arranged at this point that drainage is concurrently obtained from the stomach and jejunum and terminal ileum. This later procedure would, for example, permit the recognition of bleeding commencing in a duodenal ulcer after the balloon had reached the terminal ileum. Intubation is more useful in cases of persistent low grade bleeding than in massive intermittent bleeding.

A recent example of the use of intubation for the localization of bleeding concerns a middle aged female who had manifested occult bleeding for a known period of eight years without remission. Repeated x-ray studies had not been helpful. Small flecks of gross blood were encountered when the tube reached the lower jejunum. Exploration revealed a partial intussusception, the leading point of which was a bleeding polyp. The lesion was resected and post-operative studies have revealed no subsequent evidence of gastrointestinal bleeding.

When bleeding is demonstrated by intubation at the level of the ileocecal valve and is not present in the proximal ileum, the possibility of a bleeding Meckel's diverticulum should be strongly considered. Inasmuch as the diagnosis of such lesions is normally only a matter of suspicion and since their surgical correction is not difficult such evidence can prove most useful.

When through the foregoing procedures the source of bleeding is found to be below the ileocecal valve but superior to the highest point which can be reached by sigmoidoscopy a concerted diagnostic effort can be directed to this area. Intubation has not proved too effective in this area because of the thickened character of the intestinal contents. The radiologist, however, can make use of his best contrast techniques. If all else fails, the surgeon can, if necessary, following careful exploration inspect the inner surface of the colon with a proctoscope inserted through strategic openings. Small bleeding lesions such as hemangiomata or small polyps are often found in this way.

In conclusion the problem of gastrointestinal bleeding remains a dangerous one from the standpoint of the patient and frequently a difficult one diagnostically and therapeutically for the physician. While a small group of common lesions are responsible for the majority of cases of bleeding the less common causes are almost without number. Frequently multiple lesions exist, each of which is potentially a cause of bleeding, and each of which may best be treated differently. In the obscure case much help, both from the standpoint of exact diagnosis and planning treatment can be obtained by the accurate localization of the bleeding point. The use of the Einhorn string, combination endoscopy, and intubation for this purpose have been discussed.

BLOOD AMMONIA LEVELS IN CONGESTIVE HEART FAILURE

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DISORIENTATION, delirium, frank psychosis, tremors, and even more complicated pictures of mental and neurological abnormalities have long been noted during and as a terminal event of congestive heart failure. So frequently and for so long have these signs been noted that seldom is an explanation asked for. When the matter is questioned the traditional interpretation of these central nervous system manifestations utilizes the obvious state of hypoxia to explain the phenomena. Doubtless, the diminished oxygen supply to the various organs of the body must result in profound and complex metabolic changes contributing to nervous system changes. In spite of the reasonableness of this exposition it has been questioned of recent date. The belief that other factors contribute certain features is now more convincing.

For several years we have been impressed by the striking similarity in mental and neurological abnormalities seen in the patient in hepatic coma and the patient suffering severe congestive heart failure. Emotional irritability, confusion, delirium, agitation, flapping tremor, lethargy, and weakness are common to both conditions. Clarification of the pathogenesis of these complicating signs in severe liver failure immediately suggest application of the same explanation in congestive heart failure. Liver dysfunction and abnormalities coexisting in heart failure have already been the subject of interest for some time so the relationship seems less remote.

Both anatomical and functional alterations of the liver in patients with congestive heart failure have been reviewed by many investigators. (1, 2, 3, 4, 5) Anatomical changes include dilation of the sinusoids and narrowing of the liver cords in the central area of the lobules, necrosis of the central cells, condensation of reticulum in the degenerated central areas, marked thickening of the walls of the central and hepatic veins, as well as fibrosis of the liver with active filbroblastic proliferation. The degree of hepatic necrosis varies with the degree and severity of congestive heart failure. So called liver function tests are also deviated from the normal. (1, 2, 3, 4, 5) Bromsulfalein excretion is usually impaired. Mild elevation of serum bilirubin is common, but clinical jaundice is rare in the absence of pulmonary infarction. The urine urobilinogen is often elevated. Serum albumin, alkaline phosphatase, thrmol turbidity and cephalin cholesterol flocculation values are usually normal, however.

Innumerable metabolic abnormalities surely exist during any given episode of severe liver failure. The complexities of these deviations still largely defy actual measurements. This is to be expected in a situation, involving a structure with such broad functions as the liver. Its activity in protein metabolism has recently attracted much interest, chiefly because of the repeated observation that blood ammonia levels are usually quite elevated in liver failure and hepatic coma.(6, 7, 8, 9, 10, 11) Examination of the literature concerning this subject points up the curious fact that although the fundamental knowledge concerning the problem has been known to bio-chemists and physiologists for many years clinical application, has been attempted only recently.

It has been well established by experimental work that the liver is the principal site for the deamination of ammonia containing compounds absorbed by the intestine. Folin and Dennis(12) demonstrated that the ammonium content of the portal blood was significantly higher than that of the peripheral circulation. More than thirty years ago Mathews(13) reported that Eck fistula dogs fed a high protein diet developed the syndrome of "meat intoxication." It is now clear that this syndrome has a remarkable similarity to hepatic coma in humans. In fact, comparable demonstrations in humans with portocaval shunts have been made by McDermott and Adams,(14) and Havens and Child.(15) The supposition that high blood ammonia concentrations in the portal vein, resulting from the absorption of nitrogenous substances from the intestinal tract, by-pass the normal urea synthesizing mechanism in the liver is reasonable. It must be further theorized

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that these high levels of ammonia, now present in the systemic circulation exert a dilerterious effect upon the central nervous system producing the syndrome which we call hepatic coma.

Bessmann and Bessman(16) have suggested that high levels of ammonia in the blood result in the increased formation of glutamine from alpha keto glutarate with a decreased amount of the latter compound available in the central nervous system to take part in the Krebs Cycle. It should be reemphasized that an elevated blood ammonia level may well be only one of multiple metabolic and biochemical abnormalities occurring in hepatic failure and coma. Whether the mental and neurological changes in this syndrome can be charged directly and specifically to ammonia ion intoxication is to date still unsettled.

Sherlock and others,(17) finding elevated blood ammonia levels in patients with cirrhosis who evidenced good hepatic cellular function convincingly suggested portal systemic shunting of ammonia rich blood by and around the normally functioning liver cell. The effect upon the central nervous system, they have termed portal systemic encephalopathy. Here the situation is one of circulatory failure, after a fashion, because of abnormal collateral shunting of portal blood either around or through the liver. In the patients with acute severe hepatocellular damage, the explanation must be altered. Here the liver is so severely damaged and parenchymal cell function is at such low ebb that the liver acts as "a sieve", failing to perform its function of deamination of nitrogenous materials and passing these on into the systemic circulation.

The above mentioned mechanisms may be operating in patients with congestive heart failure. Severe liver damage resulting from congestive heart failure may interfere with the deamination of ammonia containing compounds brought to the liver by the portal vein. Increased pressure in the hepatic veins and sinusoids, as a result of congestive failure may increase portal vein pressure with a resultant increase in the flow of the ammonia rich portal blood through portocaval collateral vessels such as the gastroesophageal, hemorrhoidal and superficial abdominal veins. Thus it is seen that in congestive heart failure there are two mechanisms which may result in increased systemic blood ammonia concentrations.

The possibility that patients with congestive heart failure are prone to develop "ammonia intoxication" assumes more than academic interest. Three of the main therapeutic agents used in the treatment of these patients — ammonium chloride, cation exchange resins, and diamox have been shown to be precipitating agents in the production of hepatic coma in patients with liver disease. The possibility that these therapeutic agents may actually be more harmful than beneficial in patients with severe passive congestion of the liver due to cardiac failure must be considered.

Elevated blood ammonia levels have already been reported in patients with congestive heart failure. In a series of 9 patients Bessman and Evans(21) found elevated blood ammonia levels in eight. It is the purpose of this report to present our observations of blood ammonia levels in patients with congestive heart failure studied during the past twelve months.

Material and Methods

Fasting blood ammonia concentrations were performed in twenty-six patients who were hospitalized because of congestive heart failure. There were fifteen males and eleven females. Their average age was 56.6 years with a range from thirty-two to seventy-three years. Ten patients had arteriosclerotic heart disease, eight had rheumatic heart disease, six had hypertensive heart disease, two had cor pulmonale, two syphilitic heart disease, and one had kyphoscoliotic heart disease. Three patients had two types of heart disease.

All of the patients in this study had hepatomegaly. The degree of liver enlargement was determined by palpation and percussion. The liver was considered to be slightly enlarged if the inferior border was detectable up to four centimeters below the right costal margin in the midclavicular line. If the border was four to eight centimeters below the costal margin, the hepatomegaly was classed as moderate. Markedly enlarged livers were those whose lower borders extended over eight centimeters below the right costal margin. Two patients had slightly enlarged livers, nineteen had moderately enlarged livers and five had markedly enlarged livers. Eleven of the twenty-six patients showed some abnormality of the standard liver function tests. None of the patients had

dilated abdominal veins, hemorrhoids, esophageal varices or other evidence of portocaval collateral circulation. Abnormally high venous pressures were recorded in eleven of thirteen patients in whom they were performed.

The blood ammonia determination was carried out by a modification of Conway's method.(22) Venous blood was introduced into a saturated solution of potassium carbonate in the outer chamber of a Conway dish. The inner chamber containing 0.0002N hydrochloric acid with methyl red methylene blue indicator was titrated with 0.0005N barium hydroxide after diffusion was allowed to proceed at room temperature for fifteen minutes. A blank and a standard solution of ammonium sulfate were run simultaneously with the unknown. The details of this determination are reported elsewhere.(23) In all cases, the diffusion process was started within five minutes after the blood was shed. It has been shown that the ammonia content of the blood is stable up to twenty minutes after the blood is shed.(23)

In our laboratory normal values for fasting blood ammonia range from 50 to 110 micrograms per one hundred milliliters. Abnormal values are those above 135 micrograms per one hundred milliliters. Concentrations between 110 and 135 micrograms per one hundred milliliters are in a twilight zone and considered neither normal or abnormal by us. Figure 1 shows the percental distribution of various blood ammonia concentrations as determined by our laboratory in one hundred normal fasting persons.

Results

Table I lists the blood ammonia concentrations of the patients in this series. It will be noted that out of a total of twenty-nine blood ammonia values, twenty-five were in the normal range, two were in the twilight range (C. B. and A. M.), and two were markedly elevated (L. F. and R. P.). We were unable to obtain any significant correlation between the blood ammonia concentrations and the size of the liver, duration of congestive failure, height of venous pressure, or the degree of abnormality of the standard hepatic function tests.

Case Reports

The clinical course of the two patients in whom abnormally high blood ammonia levels were found are reported below:

TABLE I

Patient	Blood Ammonia (micrograms %)	Central Nervous System Abnormalities
O. F.	59	O
E. C.	77	O
V. T.	75	O
J. V.	62	O
D. C.	77	O
C. B.	62, 110	O
L. F.	176	X
I. M.	70	O
L. F.	71	O
T. V.	91	O
A. M.	111, 89	O
F. H.	84	O
G. H.	92	O
M. J.	62	O
M. K.	92	O
F. M.	69	O
G. F.	71	O
W. N.	82	O
C. K.	82	O
J. B.	77	O
S. H.	82	O
M. L.	92	O
G. H.	67	O
H. W.	62	O
L. N.	107	O
R. P.	164, 81	X

Case 1. L. F., a 35 year old male was admitted to the hospital with a diagnosis of subacute bacterial endocarditis due to streptococcus viridans. The patient had had rheumatic fever at the age of 12. He had been asymptomatic until 6 months prior to admission when he noted fever and fatigue. Three weeks prior to admission he developed signs and symptoms of congestive heart failure.

Physical examination revealed cardiomegaly. Murmurs of mitral stenosis and insufficiency were present. Moist rales were present in both lung bases. The liver was enlarged to 7 cm. below the right costal margin. The spleen was palpable 4 cm. below the left costal margin. Moderate ascites was noted. Two plus sacral, pretibial and pedal edema was present. Several splinter hemorrhages were noted in the nail beds.

Nonprotein nitrogen was normal. Liver function tests were within normal limits. Blood cultures positive for streptococcus viridans. Blood ammonia was 62 mocrograms %.

The patient was digitalized, treated with diuretics, and given eight million units of penicillin daily for six weeks. On this regimen, he became afebrile and recompensated. He was discharged on a low salt diet and maintenance gitalin after almost two months of hospitalization.

Seventeen days after being discharged, he was readmitted acutely ill. For three days prior to this admission the patient had noted lethargy, nausea, vomiting, anorexia, oliguria and ankle edema.

At this time he was extremely lethargic and

apathetic and unable to give a reliable history. No fever was present. The lungs were clear. The cardiac murmurs were unchanged. The liver was enlarged 8 cms. below the right costal margin. Two plus pedal edema was present. Bilateral Babinski signs were noted.

During this hospitalization the blood nonprotein nitrogen rose steadily from 114 to 155 mgm. %. The serum sodium ranged from 119 to 132 meq./L.; serum potassium from 7.0 to 7.9 meq./L., and the serum chloride from 89 to 96 meq./L. Carbon dioxide combining power varied from 8.8 to 15.4 meq./L. Hepatogram showed a bilirubin of 7.4 mgm. %, alkaline phosphatse of 31 millimol units and cholesterol esters of 11%. Blood cultures were negative. The blood ammonia was 176 micrograms %.

The patient was treated with low salt diet, gitalin, and parenteral fluids. Throughout his brief hospitalization, he was restless, confused, drowsy, irritable and hallucinating. During the last forty-eight hours of his life, he became jaundiced and comatose. He gradually went downhill and died six days after admission to the hospital.

At autopsy the heart weighed 1000 grams. The mitral valve showed an old rheumatic valvulitis with rupture of many chorda tendineae due to healing verrucae. There was dilation of the right atrium and right ventricle. The left atrium was markedly dilated and contained a mural thrombus. The lungs showed chronic passive congestion. There were tiny healing infarcts in the spleen and kidneys. The liver weighed 1200 grams and showed central necrosis. Tremendous passive congestion was present. In many places the congestion was of such a degree as to stimulate hemorrhage in the liver. The sinusoids and liver parenchymal cells were completely overwhelmed by the amount of erythocytes present. It was the conclusion of the pathologist that the superimposed subacute bacterial endocarditis, by further damaging the mitral valve, contributed to congrestive failure. It was thought that the endocarditis was healed, but unfortunately the vegetations were not cultured.

During the last few days of this patient's life, he showed many of the signs of a patient in liver failure and coma. This patient had had no liver disease until congestive heart failure developed. It is reasonable to assume that all the anatomical and functional changes in the patient's liver were due to congestive heart failure. During his first hospitalization, the patient was clear mentally and had a normal blood ammonia level. At the time of his demise, he had a markedly elevated blood ammonia level and showed mental and neurological abnormalities.

Case 2. R. P., a 59 year old man was admitted to the hospital in congestive heart failure of one month's duration manifested by dyspnea, orthopnea, and edema. He had had diabetes for the past 15 years. At the time of admission, he was taking 60 units of NPH insulin daily, digitalis, and ammonium chloride.

On admission he was disoriented, lethargic, and repetitious in his conversation. History taking was difficult, because of the patient's mental status. Coarse moist rales were present in the right lung base. The heart was not enlarged to percussion, but a grade two apical systolic murmur was present. Minimal ascites was noted. The liver edge was palpable 6 cms. below the right costal margin. Two plus pitting edema of the feet and ankles was present. The hepatogram was normal. The electrocardiogram showed an old anteroseptal myocardial infarction.

The blood ammonia concentration was 164 micrograms % on the day following admission. The patient was placed on a 1700 calorie rice and fruit diet that contained 50 mgm. of sodium a day. He was given 20 units of NPH insulin and 0.15 mgm. of digitoxin daily. His weight dropped from 160 to 139 pounds and his dyspnea, orthopnea, and edema disappeared. For the first few days in the hospital, his sensorium remained unchanged. Then it gradually became clearer as his congestive failure was controlled. On the day of his discharge from the hospital, he was mentally alert and fully oriented. A repeat blood ammonia level at that time was 81 micrograms %.

This patient showed an abnormally high blood ammonia level associated with mental abberations and cardiac decomposition with passive congestion of the liver. As he responded to treatment, his liver became smaller, the blood ammonia level returned to normal and he became mentally alert again. It is interesting to speculate on the role of the ammonium chloride that he received prior to admission in the production of a high blood ammonia level in this patient. Hepatic coma has been

produced in patients with liver disease by the administration of ammonium chloride.(24)

Discussion

In view of the autopsy findings, it seems obvious that the cause of death in the first case was severe congestive heart failure. The severe degree of congestion of the liver was sufficient to result in a marked degree of jaundice as well as to cause abnormalities in the standard liver function tests. The abnormally high blood ammonia demonstrated in this patient can best be explained by the severe degree of hepatic damage. It is postulated that some of the high ammonia containing portal vein blood was shunted around the liver and directly into the systemic circulation as a result of the tremendously increased intrahepatic pressure. In addition, the surviving hepatic cells were probably unable to synthesize urea from ammonia because of damage to them as a result of high intravascular pressure and hypoxia within the liver. We are unable to satisfactorily explain the azotemia in the patient. Possibly it may have been the result of renal dysfunction, secondary to severe chronic passive congestion and hypoxia.

The second case illustrates a correlation between congestive heart failure, elevated blood ammonia level and mental aberrations. Again we wish to emphasize that an elevated blood ammonia level is only one metabolic abnormality in a complex picture. Any attempt to explain the mental or neurological picture of hepatic coma on the basis of an elevated blood ammonia alone is a gross oversimplification. Numerous other metabolic abnormalities are undoubtedly present.

Since it has been shown repeatedly that the administration of diamox, ammonium chloride, and cation exchange resins in hepatic disease may cause an elevation of the blood ammonia level, (11, 18, 19, 20) it becomes obvious that these therapeutic agents may be contraindicated in severe chronic passive congestion of the liver. It seems apparent from clinical studies that an elevation of the blood ammonia is associated with definite neurological and mental signs and symptoms.(7, 8, 9, 10)

It is apparent from the results of this study that a great many patients with congestive heart failure and chronic passive congestion of the liver have normal blood ammonia levels. Since diamox, ammonium chloride and cation exchange resins have been used repeatedly in the routine treatment of congestive failure without detremental effects, it seems reasonable to assume that in the average case, they are relatively inocuous. However, if a patient in congestive failure shows a severe degree of hepatic congestion and/or mental or neurological aberrations, the administration of any of these agents which are known to be able to precipate hepatic coma is contraindicated. In brief, it is to be emphasized that the indiscriminate use of ammonium chloride, diamox, and cation exchange resins in congestive heart failure is to be condemned. While the use of these agents is beneficial in most patients; they may well be detrimental in some patients.

In the future, additional studies of the blood ammonia levels in patients with congestive heart failure before and after the administration of diamox, ammonia chloride, and cation exchange resins are contemplated. The use of the ammonia chloride tolerance test as described by White et al(24) in patients with liver disease would be of interest in this group of patients. The role of antibiotics in lowering an abnormally high blood ammonia by decreasing the amount of amine forming bacterial flora in the intestinal tract needs to be evaluated. The part played by electrolyte imbalance in congestive heart failure in relation to mental and neurologic changes requires further investigation.

Summary

1. Blood ammonia levels were determined in twenty-six patients with congestive heart failure and hepatomegaly. A total of twenty-nine determinations were performed of which twenty-five were normal, two border line, and two markedly elevated.

2. The clinical course of the two patients with elevated blood ammonia levels are reported. Both patients exhibited mental and neurological abnormalities similar to those found in patients in hepatic coma.

3. Possible explanations for elevated blood ammonia levels in congestive heart failure are discussed.

4. The potential hazards involved in the use of diamox, ammonium chloride and cation exchange resins in patients with congestive heart failure and hepatic damage is emphasized.

5. The need for additional investigative studies in this field is brought out.

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OMISSION

In the November 1956 *issue of Arizona Medicine on Page* 399 *a credit was omitted from the first picture appearing on that page.*

The credit should have read as follows:

We thank Mr. J. A. Rivers, Esq. of J. A. Churchill Ltd., of London, England for permission to use this illustration from Dible and Davie's PATHOLOGY, published by J. B. Lippincott Co., Phila., 1950 *(3rd Ed.) page* 750.

PHOENIX Clinical CLUB

The Case History in this discussion is selected from the Case Records of the Massachusetts General Hospital, and reprinted from the New England Journal of Medicine. The discussant under Differential Diagnosis is a member of the staff of the Massachusetts General Hospital. The other discussants are members of the Phoenix Clinical Club.

MASSACHUSETTS GENERAL HOSPITAL
PRESENTATION OF CASE

A 58 YEAR old professional man was in good health until March 4, 1953 when, while buying a ticket in a railroad depot, he suddenly fell down unconscious. He was in the company of a physician who thought the patient had collapsed due to myocardial infarction, but the pulse was of normal rhythm and not unusually rapid. There were no convulsive movements. Loss of consciousness lasted approximately ten minutes and then a clearcut dysphasia became apparent. The patient was rushed to the hospital, but when an electrocardiogram proved normal he was taken home. The dysphasia gradually cleared over a period of two hours, and except for some anxiety the patient was quite normal the rest of the day. There had been no paralysis or headache. The next ten days were uneventful, but then brief attacks of dizziness appeared occurring one to five times a day. The patient usually sat down, rested his head on his arms, and in one or two minutes pronounced the attack finished. There was no rotatory feeling but rather a peculiar sensation in the head. Following at least one attack definite aphasia, chiefly nominal, occurred for about five minutes. After these episodes had persisted for about two weeks, the patient was hospitalized on March 29.

The only additional history included an uncomplicated cholecystectomy and infectious mononucleosis in 1949. General and neurologic examinations were not significantly abnormal. The pupils were equal and reacted well to light. Fundi were not remarkable. Visual fields were full. There was no nystagmus. Ocular movements were full. There was no facial weakness. Hearing was normal. Throat and tongue were normal. Motor power, coordination, finger-nose and heel-knee tests, and sensation for pin, vibration, touch, and position were not remarkable. The patient's mind and memory were intact. Blood pressure was between 160/100 and 150/90. The blood Wassermann test was negative, sedimentation rate, 6. The blood count and urinalysis showed no abnormality. Roentgenograms of the chest and skull were normal, the pineal gland lying in the midline. An electroencephalogram was entirely normal. Liver function studies and blood chemistry including sugar and urea nitrogen were normal. An exact diagnosis could not be reached and the patient was discharged with instructions to rest.

During two months of rest (April and May) he was quite symptom-free and in June returned to his office. Soon it became apparent to his wife and co-workers that he had difficulty in naming objects and people. His memory became progressively impaired and he used notes as reminders. His wife noticed an unusual verbosity in that he harangued his clients at great length, quite unlike his usual manner. On the other hand, casual acquaintances noticed nothing amiss. The patient's usual energy was preserved and he continued to work. About the beginning of July, trembling and mild weakness of the right hand appeared, followed in a few days by dragging of the right leg. Propositional speech became progressively impaired although he could still converse readily. On July 14 his condition deteriorated rather abruptly with great weakness of the right side of the body and accompanying drowsiness. The next day he could walk only with difficulty. There had been no incontinence, headache, or convulsion. He had continued to eat well.

At this time examination showed a well-developed, well-nourished man lying quietly in bed. Spontaneous and responsive speed were almost absent and the patient seemed to comprehend nothing. There was a slight tendency to weariness, but he was wide awake and usually just gazed at the examiner when asked to put out his tongue, close his eyes, and so

on. Any attempt at answering questions – "How are you?", "Do you have pain?" – soon became puzzled jargon. He did not name the simplest of objects.

The pupils were equal and reacted to light. There was no nystagmus and ocular movements were full. The fundi including disks, retina, and vessels were not remarkable. The visual fields were difficult to assess but no hemianopsia could be demonstrated. There was clear cut weakness of the right lower face. Hearing was good and the ear drums normal. Motor power in the right upper and lower limbs was 30 to 40 per cent of normal. A conspicuous abnormality was the presence of a rhythmic, flexion-extensor tremor of the fingers and of the toes and foot on the right side. The tremor was regular at approximately four per second. The face was not involved. The tendon reflexes were brisk and approximately equal on the two sides. There seemed to be a cogwheel type of rigidity in the right limbs. The plantar response was extensor on the right, flexor on the left. Sensation could not be adequately tested, but withdrawal from pinprick was more prompt on the left. The patient could walk by himself only with great difficulty owing to the weakness of the right leg. There was no incontinence or neck stiffness. Temperature, pulse, and respiration were normal. The heart and lungs were not remarkable. Blood pressure was 160/100. Peripheral pulsations were present in both internal carotids, both anterior tibials, but only in the left posterior tibial artery. Arrangements were made to transfer the patient to a hospital in a neighboring city, but during the night of July 17 he became comatose and died.

When a 58 year old man suddenly falls unconscious on the street, the first thought of his attending physician is that he has had some sort of cardiovascular accident, either cerebral or coronary, and such was apparently the situation in the case under consideration today.

Impaired consciousness may be due to many and varied causes. Ordinary vasovagal syncope, orthostatic hypotension, and peripheral circulatory collapse such as that seen in acute blood loss, shock, etc. are common causes of short periods of loss of consciousness. Likewise direct interference with blood supply to large areas of the brain such as in thrombosis of the internal carotid artery or one of its major branches may cause unconscious states, and acute trauma to the brain such as in cases of skull fractures and concussion may account for loss of consciousness. Subdural hematoma and space occupying lesions of the brain may cause coma and frequently acute cerebral vascular accidents are ushered in by a loss of consciousness. Likewise, changes in the chemical structure of the blood such as are seen in uremia, hypoglycemia and hyper-ventilation syndrome produce states of unconsciousness as do certain toxic states or infections such as encephalitis, etc.

Such symptoms are usually not localizing but denote diffuse or general involvement of the brain. However, certain areas such as the frontal lobes and the basal ganglia, when involved, are prone to produce unconscious states. These occurrences may or not be accompanied by convulsive manifestations.

The problem in solving a neurological case such as this is twofold; namely (1) to establish the localization of the lesion and (2) the nature of the lesion. The development of aphasia in our case narrows down the localization to the left hemisphere, assuming that our subject is a right-handed individual. The problem of aphasia is a very complicated one and at the present time is in a definite state of "flux" and much confusion exists in terminology of the various types of aphasia as well as the localization of the so-called speech centers of the brain. The tendency at the present time is to include one rather large so-called speech area roughly including the "quadrilateral space of Marie" which takes in the region of the motor area of Broca and extends backward along the Sylvian fissure in the upper temporal lobe to the region of the angular gyrus. In other words, this area is bounded externally by the cortex, mesially by the internal capsule, anteriorly and posteriorly by the limits of the "Insula" or the so-called "Island of Reil." The mechanism of speech, or language, is a very complex one and involves the intelligence level of the individual and all of the senses, with subcortical pathways connecting the visual, auditory, and other higher centers to the so-called speech centers of the brain in the temporal and lower frontal lobe cortical areas. For the sake of simplicity, there are two main types of aphasia:

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[1]Posner, A. C., *et al.;* Further Observations on the Use of Tetracycline Hydrochloride in Prophylaxis and Treatment of Obstetric Infections, *Antibiotics Annual* 1954-55, pp. 594-598.

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(1) the so-called motor or "efferent" which is localized primarily in the anterior portion of the quadrilateral space, or roughly in the Broca area. In such cases, the patient cannot spontaneously utter words, may simply be able to say "yes" or "no" or to produce some interjectional speech or even be able to swear. Likewise, he cannot write or repeat words although he can comprehend the meaning of words. Such patients frequently become irritable and emotionally unstable and frustrated because they cannot express what they feel or think. (2) So-called sensory or "afferent" aphasia is localized in the posterior one-half of the quadrilateral space of Marie being somewhere in the region of the angular gyrus usually. Such patients are usually able to utter words but cannot name objects, etc. and speech is apt to be "jargon" or jumbled. The more cortical the lesion, the more distorted the speech. Jargon speech is more common in subcortial lesions; and in extensive cortical lesions the patient may be entirely mute. Nominal aphasia which this patient exhibited early in his disease is a form of sensory aphasia, as is jargon aphasia, which the patient likewise exhibited later in his disease. It can therefore be concluded that our patient had a left temporal lobe lesion occupying the posterior portion of the temporal lobe just inferior to the Sylvian fissure and extending backward to the region of the angular gyrus.

What of the other symptoms in this case? After a lapse of approximately two months from the initial signs of illness, his speech and memory became more notably impaired and there were some changes in personality noticeable only to his wife and close acquaintences. Do these changes namely of "verbosity", haranguing, etc. indicate frontal lobe pathology where we have been taught that personality changes originate, or can they be explained perhaps on the basis of some lesion in the temporal lobe? Recent studies by Drs. Gibbs and John Green, indicate that certain behavior patterns are altered by temporal lobe lesions. Nor do all such changes indicate organic brain pathology, for there is considerable evidence that emotional changes may be due to so-called threat to the ego or, in plain language, frustration due to handicaps such as speech and memory defects, etc. Further developments in this case including weakness and paralysis of the right arm and leg and the lower right face, can be explained by spread of the lesion in the overlying temporal lobe to the deeper underlying structures; namely, the internal capsule. In addition to this paralysis, we have the development of a so-called cogwheel type tremor which indicates involvement of the basal ganglia. This tremor sounds rather typical of Parkinsonian tremor which is usually attributed to pathology in the region of the corpus collosum. This is an example of the so-called "escape" or "release" motor mechanism of basal ganglia lesions. There were no demonstrable eye changes but in the presence of the patient's aphasic status, hemianopsia could very well have been missed on direct examination. Death apparently came as the result of sudden coma, approximately four and one-half months from the onset of illness.

Having localized the lesion in the left hemisphere, temporal lobe, the posterior one-half of the quadrilateral space of Marie, what is the nature of the pathological process? The causes of aphasia, which is one of the outstanding symptoms in this case, are usually organic and can be discussed under the following categories: (1) Vascular lesions, (2) Degenerative or atrophic lesions, (3) Neoplastic diseases, (4) Inflammatory diseases, and (5) Traumatic lesions. Of these groups, I feel that only vascular and neoplastic lesions deserve serious consideration. Of the so-called degenerative or atrophic conditions, mention should be made of Alzheimer's Disease which can mimic vascular disease of the brain. This disease runs a progressive course, can frequently involve the temporal lobe, produces dementia and results in a fatal outcome in 2-3 years. Progress of the disease in this instance, I believe, is too acute for this diagnosis.

Inflammatory diseases such as encephalitis, etc. are not suggested by the protocol. Neither is a metastatic abscess in the temporal lobe as the result of an extension from a middle ear or mastoid infection. Syphiloma of the brain in a temporal lobe could explain symptoms but in the absence of a positive serology and spinal fluid findings, this diagnosis cannot be entertained. (Incidentally, one cannot help wondering why spinal fluid studies were not done in this case, and likewise why more complete central nervous system studies were not done. It is assumed, of course, that the proposed move to another hospital just prior to the patient's demise was for this purpose).

Of the two remaining categories, neoplasm versus vascular disease of the brain, I feel that only the autopsy surgeon can be expected to give a competent answer and all that others of us can do, is to hazard a guess. Our man is in the age group for either cardiovascular accident or tumor. If he had a cardiovascular accident it was likely a progressive thrombosis (as are 82% of vascular lesions of the brain) of the sylvian branches of the middle cerebral artery with resultant progression of softening first of the cortical and later of the subcortial layers of the left temporal lobe with a further vascular insult probably in the deeper branches of the same artery at the time of his death. Our patient had moderate hypertension and was in the arteriosclerotic age group. Perhaps a lesion such as thromboangitis obliterans might have been the basis of his vascular pathology.

If a tumor were present, glioblastoma multiforme is the most likely choice (30% of brain tumors). This tumor is very rapidly growing and may simulate vascular disease in many instances. It occupies the cerebral hemispheres usually and often ends fatally in twelve months or less. It occurs in the middle age group, frequently undergoes cystic degeneration and frequently is complicated by necrotic changes, hemorrhage, etc. Against the diagnosis of tumor is the reported lack of shift in the pineal body (done early) and the apparent absence of signs of increased intracranial pressure, (negative eye grounds and absence of headache and vomiting). However, in certain areas of the brain such as the temporal lobe, other symptoms may indicate pressure mentioned above. There is something about this case that somehow does not ring true for a vascular accident and I therefore chose to believe that this man died from a glioblastoma originating in the left temporal lobe of the brain and finally breaking through into the internal capsule in the basal ganglia area, probably producing extremis as the result of hemorrhage or rupture of a cystic degenerated tumor into a vital area of the brain.

BEN P. FRISSELL, M.D.

DISCUSSION

At the time of the patient's hospital admission one month after onset of his illness, it was thought that cerebral vascular disease was probably responsible for his symptoms. Sudden fainting spells are not at all rare in older people who are quite well otherwise. Such attacks are commonly akinetic, convulsive movements not being part of the picture. The pathologic or pathophysiologic basis for such episodes is unknown, but cerebral arteriosclerosis is often held responsible. To have dysphasia follow such episodes is rare and some other category of attack has to be considered. In the case at hand, the sudden onset, unconsciousness, temporary dysphasia, lack of convulsive movements, the age of the patient, and the presence of mild arterial hypertension pointed strongly to vascular insult. When mild attacks of dizziness appeared, followed at least once by dysphasia, the picture was reminiscent of the transient episodes which often herald the onset of a stroke due to cerebral thrombosis. The negative medical and neurologic examinations bore out this clinical impression, which was further substantiated when rest for two months resulted in complete relief of symptoms.

When the patient was hospialized it was also suggested that he was having transient attacks of cerebral ischemia due to temporary cardiac dysfunction. Such an interpretation is often favored by cardiologists in similar cases but, in the absence of evident cardiac disease, the Stokes-Adams syndrome is, for practical purposes, the only type of cardiac disorder likely to lead to recurrent cerebral disturbances. The heart is probably much too quickly blamed for atypical vertigo, faintness, or attacks of so-called cerebral vasospasm, when a careful analysis of the patient's complaints will usually suffice to indicate the true nature of the symptoms.

After two months free of symptoms, there occurred gradually a progressive deterioration, characterized in order of appearance by nominal aphasia, memory loss, weakness of right arm and leg, drowsiness, complete aphasia, and death. The patient's relatives, although not certain, thought that these deficits appeared insidiously (until the last few days when deterioration was rapid) and had not advanced in an abrupt stuttering fashion. If this information was correct the diagnosis of incipient cerebral thrombosis would have to be revised. Cerebral thrombosis practically never produces a slowly progressive decline, but tends rather to lead to episodic, abrupt worsenings, fractions of the total hemiplegia being added from time

to time. This is true also of thrombosis of the internal carotid artery, and in this case pulsation in both carotid systems was good. Rarely, an intracerebral hemorrhage leads to increasing deficit over a period of a month or more, but usually headache is present.

If the relatives' story was reliable, the slow, progressive course would point to an expanding intracranial lesion, particularly a tumor either primary or secondary. Yet at no time was there headache; papilledema was absent the day before death, and previous roentgenograms of the skull and an electroencephalogram were normal. Furthermore, the onset of illness with a sudden attack of unconsciousness followed by spells of dizziness did not suggest tumor. In regard to secondary tumor, there was no weight loss, the sedimentation rate was normal, and a roentgenogram of the chest showed no lesion. Secondary tumor deposits in the brain rarely run their course without the appearance of pulmonary lesions, but a film of the chest had not been made in the weeks prior to death. An intracranial abcess occasionally occurs unexpectedly without any source and unassociated with signs of meningeal irritation, but in this case the illness had been too lengthy. The presence of a subdural hematoma had to be kept in mind, although the slow, progressive type will not lead to total aphasia without marked disturbance of consciousness. There was no history of injury to the head but, of course, this is not significant. The abrupt onset of the first symptoms and the lack of headache would be atypical of a subdural hematoma.

The presence of the rhythmic, four per second tremor of the right hand and foot was most striking, but unfortunately no one who was consulted was aware of any diagnostic value attached to such a sign. The tremor was clearly not that of partial continual epilepsy but mimicked the trembling of parkinsonism, except for its slightly faster rate. The fingers and toes seemed to flex synchronously and all five digits of the hand were involved.

Finally, the diagnosis of suspected brain tumor was made. In this conclusion, the character of the onset, the period of cessation of symptoms, and the absence of headache and papilledema were disregarded, in view of the slow, progressive final course which is scarcely ever due to vascular disease.

PATHOLOGIC FINDINGS

The left temporal lobe, from its anterior tip to a point 7 cm. posterior was occupied or replaced by a huge tumor mass, which histologically was of the glioblastoma multiforme type. A large uncal herniation on the left side was associated with multiple pontine hemorrhages. In its deeper portion the tumor clearly invaded the inferolateral portion of the putamen and adjacent internal capsule.

COMMENT

This case is an example of an extensive tumor running its entire course without headache or pailledema. The interpretation of the progressive course as suggestive of tumor rather than arterial disease proved correct. Pathogenesis of the initial attack remains obscure, but the attacks of dizziness probably were minor seizures. The failure of the electro-enchephalogram to show an abnormality provides another example of the fact that as a rule clinical data must still provide the essential ingredients of a correct diagnosis. The nature of the rhythmic tremor of the paretic limbs remains obscure, but since it is most unusual for vascular disease to produce such a picture, it is possible that the presence of a tremor might be indicative of tumor.

THE President's PAGE

ARIZONA MEDICAL ASSOCIATION

RECENTLY, the Council has made several important changes at the central office of the Arizona Medical Association. It is important that you should know about these changes, and I am sure that you will approve of them, as I believe that they will increase the efficiency in the management of the affairs of the Association.

The Council has employed Paul Boykin as assistant Executive Secretary, to assist our able Bob Carpenter. Mr. Boykin will relieve Bob of the many routine duties that have taken up a major part of his time and energy. This will permit Bob to concentrate on the weightier problems, and allow him some time to travel around the state and to meet more often with the county societies and their members. Every member should meet Mr. Carpenter, – for he is not only a valuable, tireless, worker, but a very personable man also. Paul Boykin gives promise of being another valuable addition to our staff.

The other major change initiated by the council has been the removal of our office to 826 Security Building in Phoenix. These larger quarters provide adequate space for our ever-growing files. The space is shared, as is the expense, with the Arizona Board of Medical Examiners.

As an officer of the Association and member of the Council for over 10 years, I recognize the tremendous volume of business that is entailed in the management of the affairs of our Medical Association. The volume of incoming and out-going mail, telephone and telegraph messages is tremendous. Consider just the matter of transposing, typing or mimeographing the minutes of the meetings of the Council, House of Delegates, and the various Boards, committees and sub-committees; there are 27 major committees, and they meet several times a year. The Council meets at least 5 or 6 times a year, – probably more often. There is rarely a Saturday or Sunday during the year that is without an important meeting.

When the Legislature is in session, greater demands are made upon the energies and time of our personnel. They are also well-informed of the progress of legislation in Washington. Close contact is also maintained with the A.M.A. headquarters in Chicago, and with the Washington office of the A.M.A.

I have been able to point out to you but a few of the services performed by our loyal staff at the office of the Arizona Medical Association. I invite you to visit our new quarters at 826 Security Building and meet the competent people who serve you. I assure you that your visit will be rewarding to yourself, and appreciated by Bob, Paul, and Mrs. Olson.

Next month, I hope to be able to report to you on the results of our negotiations on the Medicare Program.

A. I. PODOLSKY, M.D., President
Arizona Medical Association, Inc.

Editorial

ARIZONA MEDICINE

Journal of
ARIZONA MEDICAL ASSOCIATION, INC.

VOL. 13 NOVEMBER, 1956 NO. 11

CONTRIBUTORS

The Editor sincerely solicits contributions of scientific articles for publication in ARIZONA MEDICINE. All such contributions are greatly appreciated. All will be given equal consideration.

Certain general rules must be followed, however, and the Editor therefore respectfully submits the following suggestions to authors and contributors:

1. Follow the general rules of good English, especially with regard to construction, diction, spelling, and punctuation.
2. Be guided by the general rules of medical writing as followed by the JOURNAL OF THE AMERICAN MEDICAL ASSOCIATION.
3. Be brief, even while being thorough and complete. Avoid unnecessary words. Try to limit the article to 1500 words.
4. Read and re-read the manuscript several times to correct it, especially for spelling and punctuation.
5. Manuscripts should be typewritten, double spaced, and the original and a carbon copy submitted.
6. Articles for publication should have been read before a controversial body, e.g., a hospital staff meeting, or a county medical society meeting.
7. Exclusive Publication—Articles are accepted for publication on condition that they are contributed solely to this Journal. Ordinarily contributors will be notifed within 60 days if a manuscript is accepted for publication. Every effort will be made to return unused manuscripts.
8. Illustrations — Ordinarily publication of 2 or 3 illustrations accompanying an article will be paid for by Arizona Medicine. Any number beyond this will have to be paid for by the author.
9. Reprints — Reprints must be paid for by the author at established standard rates.

The Editor is always ready, willing, and happy to help in any way possible.

POLICY

EFFORTS are being made by the Editorial Board to emphasize the medical-legal, medical-economic and medical-social aspects more heavily than they have been in the best. Undoubtedly this policy will precipitate considerably controversy. This is welcomed and the editors of the Journal would appreciate receiving letters to the Editor which can bring out various viewpoints and be published in later issues of the Journal. We feel that one purpose this Journal can serve is to present problems that are unique to medical practice in the Southwest. Adequate knowledge and discussion of these problems should prove helpful.

TOBACCO AND CANCER

THERE has been much written about the effects of tobacco on males and females alike and a great many theories offered about the problem of smoking and carinoma of the respiratory system. Most of the comments in the literature have been relative to the association or correlation between smoking and bronchogenic carcinoma. Not long after the initial comments about smoking and carcinoma of the lungs were made, there was an adverse effect on the tobacco industry and the consumption of cigarettes. Stocks in several large companies dropped several points and consumption of cigarettes was estimated to be off about ten per cent at one time. However, during the last year or so cigarette consumption again has risen and surpassed previous high figures.

Such a large industry naturally would not accept only words regarding such serious accusations but demanded scientific proof. Exact scientific proof that carcinogenic agents were present in cigarettes or tobacco smoking was not forthcoming, at least not up to the present time. However, there have been indications from certain laboratories that certain derivatives from tobacco may act as carcinogenic agents.

In rebuttal the tobacco industry has not been idle but has formed a Tobacco Industry Research Committee and only recently a pre-

liminary report was issued under the editorship of the scientific director Clarence Cook Little. The Tobacco Industry Research Committee (T.I.R.C.) has made many monetary grants distributed in eighteen states throughout the nation, to various institutions, and some fifty-five scientists who with their colleagues are participating in a carefully planned, well integrated scientific endeavor to help in solving of important health questions.

As of August 1956 the research grants distributed by the committee amounted to $1,500,000. It is organized with a Scientific Advisory Board which comprises many illustrious names in various phases of medicine and related sciences. The sole purpose of the T.I.R.C. is to encourage and support qualified research scientists in their effort to learn more about such complex problems as heart disease and cancer, meanwhile refraining from premature judgments.

Three scientific areas of study or interest stressed in their report are lung tissue study, studies in heart circulation, and research in tissue culture. Also, the committee and the advisory board have set out to promote the development of acceptable standards governing work with tobacco derivatives to meet a challenging need among investigators. Their contention is that many experiments with tobacco smoke constituents have been or were being conducted under conditions bearing little if any relation to human use of tobacco. A defined range of methods and conditions was needed to give a greater degree of pertinency of the experiment to the experience of human smoking. To date substantial progress has been made in (1) preparation of standard tobacco derivatives for laboratory use, (2) definition of standard operating conditions for smoking machines, including control of temperature ranges in subjecting material to combustion, (3) further development and production of genetically controlled laboratory animals, particularly mice, with known characteristics, to be used in tobacco experiments.

The T.I.R.C. desires to have scientists work with the greatest freedom and without domination of any kind. It claims that it will make no attempt to direct the administration of the projects once started, to influence its course or to control its results other than to be assured that the funds are properly expended for the purposes of the grant and that all findings are reported in accordance with the best scientific practices.

It would appear from the preliminary report of the T.I.R.C. that it exhibits a willingness to finance research in our health problems under the guidance of a recognized scientific advisory board. Whatever the outcome may be, the initial approach is certainly to be commended when it is recognized the public is not to be subjected to many rebuttals and unscientific claims by the tobacco industry in answer to the numerous scientific articles published against it.

C.T.R.

ORAL TREATMENT OF DIABETES

PRELIMINARY tests of the hypoglycemic sulfonamide deriviates indicated that these drugs might be effective in the treatment of diabetes mellitus, when given orally. The drugs (BZ-55 and Orinase) were soon found to be of little or no value in the management of the severe, brittle or juvenile types. The mode of action of these sulfonamides is still in doubt.

Even though these drugs may produce hypoglycemia in some subjects, it must be remembered that the simple normalizing of the blood sugar of a diabetic does not necessarily mean that the diabetes is controlled in toto. Because the complications of diabetes usually develop slowly, it will take many years, 15 or 20, to determine the effectiveness of any therapeutic antidiabetic agent.

Past experience with the sulfonamides has taught that those drugs are potentially toxic. Even though toxic reactions from these hypoglycemic-sulfonamides were absent or rare in early studies, as time has passed and their use widened, toxic reactions are becoming manifest. In some instances these reactions are very serious and possibly contributory to death. It is now known that these toxic reactions are not infrequent.

Insulin remains our best lifesaving therapeutic agent for the treatment of diabetes mellitus. Other drugs for the control of diabetes must remain in the hands of qualified research teams, to be proved by years of study.

L.B.S.

COUNCIL ACTION

COUNCIL of the Arizona Medical Association met in Phoenix September 23rd from 11:00 A.M. to 4:45 P.M. Among actions taken was approval of the supplemental insurance benefits in the Association-sponsored group sickness and accident policy carried by the National Casualty Company of Detroit. Individual contacts are to be made by the Arizona underwriters of this policy. Up to $500.00 per month benefits for five years is now available to members of the Association, by taking advantage of basic coverage, extended coverage, and supplemental coverage.

The Medicare Program for medical care of military dependents by civilian physicians and hospitals (under certain prescribed circumstances) was discussed at great length by Council. A state-wide contract between the defense department and physicians is planned. A fee schedule committee was appointed, composed of the following members, Dr. Frank W. Edell, chairman:

Ernest A. Born, M.D.
Carlos C. Craig, M. D.
Hugh E. Dierker, M.D.
John A. Eisenbeiss, M.D.
Francis M. Findlay, M.D.
George O. Hartman, M.D.
Robert E. Hastings, M.D.
Benjamin Hertzberg, M.D.
Walter T. Hileman, M.D.
Paul B. Jarrett, M.D.
Elvie B. Jolley, M.D.
Harry B. Lehmberg, M.D.
Donald E. Nelson, M.D.
Royal W. Rudolph, M.D.
E. Henry Running, M.D.
Stuart Sanger, M.D.
Paul L. Singer, M.D.
William B. Steen, M.D.
Oscar W. Thoeny, M.D.
Otto E. Utzinger, M.D.
Charles E. Van Epps, M.D.
James Volpe, M.D.
MacDonald Wood, M.D.

Council designated Blue Shield as the fiscal agent for the first year of operation under Medicare. This is a bookkeeping function only.

The fee schedule committee met in Phoenix on Sunday, October 7th and arrived at a fee schedule for negotiation with the defense department. This fee schedule is based on the California relative value schedule as adopted by the Council of the California Medical Association on February 12, 1956.

The fee schedule recommended by the committee will be presented to the defense department in Washington later this year for final negotiation on a state-wide basis. December 8th is the target date for beginning operation of the Medicare Plan, as authorized by public law 569 passed by the last congress and signed by President Eisenhower on June 8th.

The medical economics committee along with those from other western state associations met in Denver with the task force from the department of defense on August 25th and 26th. This committee was expanded by council to the larger commtitee above. More details will be available to membership of the Association at a later date.

TOPICS OF *Current Medical* INTEREST

RX., DX., AND DRS.

By Guillermo Osler, M.D.

SOME day you may be invited to speak to a HIGH SCHOOL CLUB. We warn you, in case you haven't known it, that the sponsor may be a group of girls who are aiming at a NURSING career, or a group of boys who intend to be DOCTORS. . . . They really are organized that early, and the result might someday be nurses and medics who are better (and sooner) prepared to face the decisions which have to be made at the time of graduation. . . . Put on your best clothes and manners, and tell them your best jokes if not the truth.

Kirby, Corpron, and Tanner use an uncommon word to call attention to 'hospital-acquired' infections, — NOSOCOMIAL. They may be serious, and deserve serious attention. . . . They stress their finding that coliform bacteria may cause infections in hospitals, in addition to those by staphylococcus. (M.pyogenes). The coliform infections may involve the urinary tract, are often antibiotic-resistant, and the problem is generally unrecognized. . . . ARIZONA MEDICINE now joins the J.A.M.A. in recognizing it.

A survey of reports at the INTERNATIONAL PEDIATRIC CONGRESS by Seligmann and Levine provides some large, wonderful trends at which we can wonder. . . . The only important contagious disease threat is polio, and you've heard of the threats to polio. The hazardous diseases of 1 or 2 generations ago (scarlet fever, measles, whooping cough, et al) are largely under control. Ear infections rarely become serious. The children with diabetes, TB, heart defects and blood diseases of the newborn have only to be recognized as such to have a good to excellent chance of control or healing. . . . The problems which remain include prevention of prematurity and birth defects, prevention and treatment of cerebral palsy, muscular dystrophy, mental retardation, leukemia, cancer, allergy, and certain renal diseases. . . . Now if we can just "keep the peace" and stay prosperous we can solve a few more of these puzzles and spread medical care around a bit more.

F. A. Simmons of Boston and Harvard reaches a few interesting conclusions in "Recent Advances in INFERTILITY", (an article which was published by the MEDICAL ANNALS of the District of Columbia). . . . 'Recent advances', he says, depend on how well one keeps up with the literature. There is a rapidly increasing number of meetings on the topic of sterility. The widely published stories in the lay press about the good effects of testosterone are baseless. About 4% show good results, not 85 or 90%. The English have reported very good results in several small series from the surgical correction of varicoceles. Thirty or 40% achieve paternity.

The para-medical journal 'HOSPITALS' contained a wonderful story last month. In 1933 Dr. James Mackintosh of Scotland wrote himself a letter, to be opened when he became 65 years of age. In the years since then he has become a leading health educator, has often travelled to the U. S., and has been one of the greatest authorities on preventive medicine and hospital administration. . . . He opened the letter to himself in February 1955, and found that he had solemnly given himself several pieces of friendly advice. He urged his older self that he not become talkative, since it prevents learning; that he listen, really listen, and not simply look like it; that he not become suspicious of people's motives; that he avoid prying into the affairs of young people; that he give advice only when asked; that he not try to attract sympathy to himself; and that he not cling to his job after 65 like a barnacle, excusing it by the need to "see the job thru." . . . Dr. Mackintosh then wrote a reply, after he got the letter, to his younger self. He was grateful for the advice; he will try to continue to learn; and he had promptly retired from his job!

Upjohn's "Scope Weekly" tells the story of Johnny Longden, a man who rides horses, and how he keeps riding at the age of 43 years. A hormone does it, says the article. . . . A year ago he was fighting his weight, as well as a wide-spread "bursitis." A San Francisco urologist decided to try out a PARATHYROID hormone. The result was a clearance of the discomfort, plus a strange dividend, — the loss of his weight control problem. . . . Longdon can thank the man for fixing him up so he broke the world's record for winners (4870), and maybe fixed him up so you won $12.00 on one of his horses at Del Mar this year (tho it probably wasn't Longdon if the price was $12.00). . . . The only weakness in this story might be the size of the doctor's series. The only other person he used parathyroid on was his office nurse.

PLASMAPHARESIS consists of removing venous blood from a patient, separating plasma from cells, and returning the cells to the body.

Stokes and Smolens of the University of Pennsylvania have found that repeated plasmapharesis on a donor (as often as once a week) results in a stimulation of ANTIBODY PRODUCTION up to a very high titer. These can be used for diagnosis or therapy. . . . They predict that the method could be useful in quite a number of infections. Human tetanus antitoxin is much more effective than that from horses, and the foreign protein reactions would be almost eliminated. Etc.

Dr. Jim Perkins of the NAT'L. TB ASS'N. sums up the current statistics on the disease, and the picture is better but not pretty. . . . 1,200,000 cases of TB in the U. S. . . . 800,000 are active and infectious. . . . 250,000 or more are not under treatment. . . . 80,000 new active cases are being reported per year. . . . 16,000 persons died of the disease a year ago. . . . The disease is a hazard. It is preventable. It is not under control.

Taylor County, Texas, has started a program to do MASS ELECTROCARDIOGRARMS on everyone. They began with a survey of all employees on the Abilene 'Reporter News'. . . . The county Heart Association is in back of the drive. They hope to make it "as commonplace as TB screening," but we hope it will be more so. TB screening is far from widespread, in the first place, even tho the disease is a hazard to more than the possessor. On the other hand, cardiovascular diseases top the list of morbidity and mortality statistics, so the procedure is a logical one. . . . We can only hope that some cardiologists don't fight the use of ECGs as some radiologists fight case-finding films. One attitude would be as silly as the other, both from the dollars and 'sense' standpoint.

This pathology-etiology item may not be world-shaking, but it is quite amazing in a small way. . . . The CAUSE OF DEATH IN LEPROSY should most often be toxicity, or pneumonia, or some other infection. It isn't. . . . They say it is quite commonly amyloidosis, and the reason (just as for amyloid deposit in other situations) is uncertain.

We have picked the brains of three 'arctic-type' scientists who spoke at a Trudeau Society meeting in Los Angeles on HYPOTHERMIA, or 'cooling,' in surgery. They were Drs. (John C.) Jones, Zinn, and Warnock. . . . The chief aim of 'cooling' is to slow down the metabolism and decrease the need for oxygen. . . . The methods have included cooling the blood, flooding a pleural cavity with a cool solution, wrapping the patient in coils, and packing the patient in ice. The last-named is apparently best. . . . The temperature is lowered to a level between 31 and 16 degrees C. (87-80F). The exact level depends on the need. The patient is slowly cooled (1 degree per 15 minutes) to the proper level by anterior and posterior application of ice; he is removed from the pack before surgery; the temp. 'drifts' about 4 degrees lower after removal from the ice; he stays cold for 5 to 9 hours after unpacking. . . . No sedation or premedication is used before cooling, but a needle is put into an ankle vein for later entrance of fluids and drugs; a nitrous oxide and oxygen anaesthesia is started; curare is given for intubation; and the patient is usually digitalized. A thermo-couple is placed in the rectum. . . . As 'cooling' occurs the patient becomes very pale, the pupils dilate, the jaw sets, the pulse slows, and the audible blood pressure goes, tho fluctuation on the dial persists and the level is constant. The patient shivers at a certain level, and the temp. is carried just below that level. Shivering is a warning mechanism, and is also obvious on the ECG. . . . Shock is hard to recognize, so that excess transfusion is wise. No post-op, warmup is used, since one must avoid chillblains, skin-burns, etc. . . . The uses are diverse, and include numerous non-cardiac indications. Cardio-vascular reasons for hypothermia include cyanotic bad-risk infants, septal defects, Tetralogy of Fallot, thoracic aneurysm, vascular stenosis (Leriche syndrome), etc. . . . The procedure will allow as long as 1½ to 2 hours, with the brain or kidney blood supply shut off. The maximum time for heart surgery, however, is 7 to 12 minutes. . . . The hazards include poor control, and a skilled team is necessary for cooling, anaesthesia, and surgery. Ventricular fibrillation is the most dreaded post-op. hazard; potassium chloride is used to produce a cardiac standstill then normal rhythm is induced. Post-op. bleeding may occur; chillblains, perhaps due to fat damage, may follow; and a neuritis is occasionally seen. . . . The method is actually still under investigation, with blood chemistry being studied, attempts to find theories to fit the facts, and attempts to minimize the hazards.

SUPPLEMENTAL SICKNESS AND ACCIDENT BENEFITS

COUNCIL and the medical economics committee are considering plans for additional group sickness and accident insurance benefits, with a view to providing a total of five hundred dollars per month income for disability from sickness or accident.

In April, the medical economics committees recommended to council that this move be undertaken. On April 25, council directed that the membership be polled. (The costs were borne by the National Casualty Co.) One-third of the members replied; two-thirds of those replying favored obtaining this additional benefit. More details will be available when further plans are concluded, and will be published in the Journal.

NEW

ATARAXOID is a unique, new combination of STERANE and ATARAX, which now permits simultaneous symptomatic control and reduction of attendant anxiety and apprehension in rheumatoid arthritis and other indications.

The added tranquilizer control, desirably easing mental stress, also directly assists clinical progress. It minimizes the chance of exacerbation related to emotional strain and facilitates patient confidence and cooperation in the therapeutic program toward maximum rehabilitation.

ATARAXOID exerts the anti-rheumatic, anti-inflammatory activity of STERANE distinctly superior to previous steroids, effective in radically reduced dosage, and with minimal disturbance of electrolyte and fluid metabolism.

The ataractic effect is a central neuro-relaxing action – the result of a marked cerebral specificity – free of mental fogging and devoid of any major complications: no liver, blood or brain damage. This peace-of-mind component is also used in the lowest dosage range.

Supplied: Each green, scored, ATARAXOID oral tablet contains 5 mg. prednisolone (STERANE) and 10 mg. hydroxyzine hydrochloride (ATARAX). Bottles of 30 and 100.

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Division, Chas. Pfizer & Co., Inc.
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the first and only ataraxic-corticoid

Taraxoid*

prednisolone and hydroxyzine

Pfizer

combining the newest, safest tranquilizer, ATARAX* + the newest, most effective steroid, STERANE* (prednisolone)

simultaneously controls the symptoms and the apprehension

In Rheumatoid Arthritis, other collagen diseases, bronchial asthma and inflammatory dermatoses

*Trademark

The History of Medicine in Arizona

GEORGE EMERY GOODFELLOW, M.D.

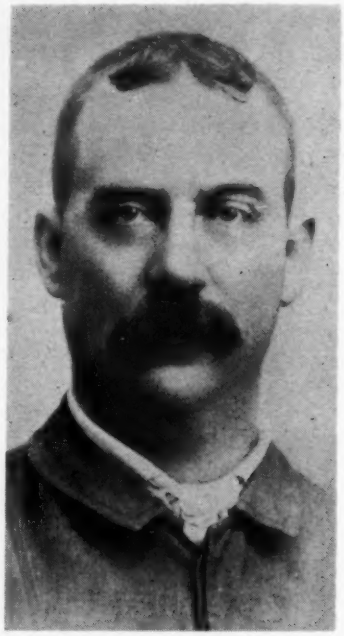
(Tombstone 1880-1891)
Tucson 1891-1898

DR. GOODFELLOW was born December 23, 1855 at Downieville, Sierra County, California. He was the son of Milton J. and Anna Amanda (Baskin) Goodfellow — both parents being natives of Pennsylvania. His father was a prominent mining engineer, who had studied both medicine and dentistry. His mother was the daughter of Dr. John Baskin — a well-known physician of Meadville.

In 1865 the family moved from Downieville to Austin, Nevada. During 1868-1870 George lived with an aunt at Meadville and attended school. Then for one year he attended the California Military Academy at Oakland. Later for one year he was a student at the University of California in the Civil Engineering Department. He declined an appointment to West Point, but later accepted the appointment, from Nevada, as Midshipman to the United States Naval Academy at Annapolis, entering June 12, 1872. Here he was the champion boxer. But on December 12, 1872 he was expelled for the hazing of a Negro cadet. The Negro seems to have been responsible for the encounter and he was himself dismissed a few months later. From 1872-1876 he was a student at the Medical Department of the University of Wooster, Cleveland, Ohio, graduating February 23, 1876. A few months later he was married to Miss Katherine Colt of Meadville. In November of that year they went to Oakland, California. In a short time his father suggested he come to Prescott, Arizona, to be the doctor of a mine of which the father was in charge. He was in Prescott for about 2 years. For a short time he was an acting assistant surgeon at Whipple Barracks. But in 1879 he again became a "contract surgeon" and was stationed at Fort Lowell. On September 15, 1880, he resigned to engage in private practice at Tombstone.

TOMBSTONE 1880-1891

All this becomes pertinent as bearing out Dr. Goodfellow's statement, viz., that he had presumably had a greater practice in gun-shot wounds of the abdomen than any other man in civil life in the country. I think we may also



George Emery Goodfellow, M.D.

conclude that this extensive practice laid the proper foundation, both in experience and courage, for him later to attempt operations — new to himself and everyone else.

Leading surgeon — During his residence in Tombstone Dr. Goodfellow was the outstanding surgeon of Arizona.

Early residents of Cochise County still relate the case of a young man who was "mad" — roaming wild and nude in the mountains. His friends took him to Tombstone where Dr. Goodfellow operated upon his skull. In a short time he returned home "clothed and in his right mind."

Dr. Goodfellow was also a pioneer in plastic surgery. In June 1881 there was a disastrous fire in Tombstone. Mr. George W. Parsons of Los Angeles writes of his experience: "I was knocked senseless by a dislodged beam and a large splinter had entered just under the skin glancing upward and just missing the eye, face quite flattened and nose all over it. Dr. Goodfellow made a plaster cast, cut away the deformity in the cast and then cut my nose loose from the bone and tacked it up in place

so that the cast, with the aid of a wire run through my nose, held it in place. I eventually recovered emerging with a fine Roman nose, free from disfigurement."

Coroner – For the 10 years Dr. Goodfellow also served as coroner. Some of his acts in that capacity brought him as much notoriety as his surgical ability did fame. Possibly the most notable – quoted in all the newspapers in the country – was the following: At 8:30 A.M. February 22, 1884, John Heath – a murderer – was lynched on Toughnut St., Tomstone. Dr. Goodfellow, as a coroner, was accused of preparing the verdict of the jury: "We the coroner's jury of Tombstone, Cochise County, Arizona, find that the deceased came to this death from emphysema of the lungs; a disease common in high altitudes; characterized by an excess of air in the cellular spaces; due to strangulation or otherwise."

Earthquake – On May 3, 1887, there was a severe earthquake in Bavispe, Sonora, Mexico and Dr. Goodfellow took a relief party of the Geologic Survey into the district a month later. They visited regions where the natives had never seen a white man or a horse and wagon. None of the injured had received aid. As a result of his surgical work Dr. Goodfellow was considered by them as a saint and was thereafter called by them "el Santo Doctor". In appreciation of this service, a horse, Kentucky bred, blooded and gaited, was presented to Dr. Goodfellow with due formality and ceremony by a small body of Mexican soldiers sent from Mexico City by President Diaz, who later became one of Goodfellow's close friends.

TUCSON 1891-1896

When Dr. Goodfellow moved to Tucson, October 23, 1891, my friend – Dr. I. B. Hamilton, of Los Angeles – succeeded him at Tombstone. About 5 months later Hamilton wrote me (Dr. Bledsoe) that he was planning to join Goodfellow here and asked me to take his place in Tombstone. After 2 weeks of correspondence he decided to remain there and I came here as assistant to Goodfellow April 20, 1892.

Southern Pacific Surgeon – Before his removal to Tucson Dr. Goodfellow had accepted the position of Railroad Division Surgeon – successor to Dr. Handy. He held this position till he removed from the Territory in 1896. Though the railroad men might cuss at not being able to find him the minute they wanted him, no one ever questioned his ability.

Of my six months' close association with Dr. Goodfellow four things stand out conspiciously.

Surgical week – At the end of my first month here Dr. Goodfellow had a "Surgical Week". He had been saving up cases from Tomstone, Bisbee, and Tucson. He had his friend – Dr. Francis Haynes, of Los Angeles – come here for a week and two major operations were performed each day. This was more operative surgery of this kind than Tucson had seen in its two centuries of existence.

Conducted Hospital Here – During his residence in Tucson Dr. Goodfellow purchased the property now known as the Orndorff Hotel and for about a year conducted it as a hospital, doing his surgical work there.

Arizona Health Officer – From 1893-1896 Dr. Goodfellow served as Territorial Health Officer, under Gov. L. C. Hughes, and everybody agreed that no more competent man could have been chosen.

First Appendectomy in Arizona – Dr. Goodfellow is credited with having performed the first appendectomy in Arizona. I know of no man so likely to have had this distinction, but have no knowledge of the date.

A PIONEER IN PROSTATECTOMY

It was while Dr. Goodfellow was in Arizona that he did pioneer work in this operation.

Initial Case – He said: "In 1891 I made a pure perineal prostatectomy, the first as far as known to me deliberately devised and carried out." He places the date as in the week of September 29, 1891, which would be during his last month in Tombstone. If this be true, then he was the first man in the world to perform this operation. This initial case, with subsequent ones, was to be reported at a meeting of the Southern California Medical Society December 3, 1896. But Dr. Goodfellow was in New York at the time and the paper was not presented or published. These early cases were not reported till 1901. Later all his records were destroyed in the San Francisco fire. So some have questioned his priority.

Testimony of Others – (1) At the meeting of the Arizona Medical Association held in Tucson in 1921 the visiting physicians attended the regular Chamber of Commerce luncheon.

When I introduced Dr. Cecil, well-known urologist of Los Angeles, his first words were an expression of his pleasure at being in Tucson, the home of Goodfellow, the father of prostatectomy.

(2) Dr. Hugh H. Young — for many years the leading authority in the United States upon this subject — not only accepted Goodfellow's claim of originating perineal prostatectomy by midline incision but states: "Goodfellow deserves the credit of being the first to make a success of prostatectomy."

Early Prostatectomy Cases — Dr. Goodfellow performed several early operations of this kind in Tucson. Some of them were notable both because it was pioneer work in a new field and because of the prominence of the patients. In several such I gave the anesthetic.

The first case — early in 1893 — was Mr. E. B. Gage — a prominent mining man of Tombstone, Dr. Goodfellow used the scalpel only to get through the skin and perineal muscle. All further dissection up to the gland and its enucleation were done by the index finger. In a remarkably short time the gland was delivered intact. It was just about the size of a chestnut and of normal pink color. A very few weeks after the operation I met Mr. Gage in the corridor of the hospital and he expressed his delight at the beneficial results of the operation.

Mr. Gage had a friend, Eames — a prominent lawyer of Chicago — who soon came here for the operation. Conditions were quite different from the case of Mr. Gage. The gland was at least two and one-half times larger, dark red in color and quite friable — about one-tenth or one-eighth coming away piece-meal.

RECORD IN SPANISH-AMERICAN WAR

Title and Authority — At the outbreak of the Spanish-American War, when his friend General William R. Shafter called he gave up his practice and went to his aid. Army officers have denied that he had any standing in the army and at the Dodge Commission investigation referred to him as "a civilian with no rank or authority". But no one else ever questioned his position.

Interpreter — He spoke Spanish fluently — better than the official interpreters. Possibly this qualification led to his being chosen envoy or intermediary for the army. During the two weeks between the battle of San Juan and El Caney (July 3) and the surrender of General Toral (July 17) time and again, blindfolded and under a flag of true, he was taken to the Spanish camp, and many, in a position to know, have given him chief credit for the surrender of Santiago de Cuba.

Citation — His services were officially recognized in the following citation (February 30, 1900): "July 1898 Dr. G. Goodfellow, civilian and volunteer aid to General William R. Shafter, for especially meritorious services, professional and military during the campaign in Cuba."

SAN FRANCISCO 1900-1907

Upon his return from the Spanish-American War Dr. Goodfellow located in San Francisco, remaining there for seven years. During this time he devoted himself exclusively to his specialty — prostatectomy. He had made a "triumphal tour" across the country, demonstrating his operation at numerous surgical centers — thus becoming a national figure. In 1902 he was guest speaker before the California Academy of Medicine and presented a paper upon his specialty. In 1904 he reported 78 perineal prostatectomies on patients ranging from 45 to 84 years of age with only two deaths, one from sepsis and the other shock.

GUAYMAS 1907-1910

In 1907 Colonel Randolph appointed Goodfellow Chief Surgeon of the Southern Pacific of Mexico, with headquarters at Guaymas. During the first year in his new position offices were opened in Tucson for the care of local employees, and he was an occasional visitor here and renewed his acquaintance with former friends.

He remained at Guaymas till 1910. In March of that year he developed multiple neuritis, suffering excrutiating pain. He was removed to a hospital in Los Angeles. In April wrist-drop occurred. He then told friends if he was doomed to be a cripple and unable to operate, he did not care to get well. During the months of his final illness he received most attentive care from the late Dr. C. W. Fish — for many years his brother-in-law, later his son-in-law. He died December 7, 1910 at Los Angeles.

Thus passed not only the most brilliant and versatile surgeon who ever practiced in Arizona but also one of the picturesque characters of Arizona's early history.

PUBLICATIONS

During a period of nearly 30 years (1879-1907) about one dozen articles of his appeared in the leading medical and surgical journals of the country. About half of them were upon some phase of his specialty – prostatectomy. A few of the earlier ones were strictly medical and the last: "The Gila Monster Again" appeared in the Scientific American. As the majority of his articles were reports of his own work, they were accepted as authoritative.

ANTIDOTE AGAINST NERVE GAS

DEVELOPMENT of the first effective **antidote** against wartime use of deadly **nerve** gas, was announced March 13 by a team of Columbia University biochemists. The antidote has protected 100 percent of the animals exposed to doses of nerve gas that would have killed them in less than five minutes. The new compound was developed and synthesized by a group headed by **Dr. Irwin B. Wilson** at Columbia's College of Physicians and Surgeons.

The chemical is named **2-pyridine aldoxime methiodine** – or PAM for short. It is a crystalline solid that dissolves in water to yield a clear yellow solution, and was reported to be "easily and inexpensively synthesized," and non-toxic in amounts necessary to preserve life. (details) (William L. Laurence – N. Y. Times 3/14 1249-04)

TRANQUILIZING DRUGS

THE FOLLOWING exerpts are reprinted from a recent bulletin on the use of tranquilizing drugs published by the APA's Committees on Research Therapy and Public Information. It is so timely that it is reprinted here.

"The profession - - - recognizes with enthusiasm the development of the tranquilizing and other drugs for the treatment of psychiatric disorders over the past four years. It seems clear that the drugs are useful adjuncts in the psychiatric treatment of certain patients in private practice and on an outpatient basis in clinics and hospitals, though the extent and the conditions under which this practice will prove sound remains to be confirmed through prolonged and careful study.

"Psychiatrists are at the same time concerned about the apparently widespread use of the drugs by the public for the relief of common anxiety, emotional upsets, nervousness, and the routine tensions of everyday living. It is reported that 35,000,000 prescriptions for the drugs will be written in 1956, and a market research firm states that 3 of 10 compounds prescribed most frequently by physicians in 1955 were tranquilizers.) Numerous articles in the public prints may be pointed to as evidence that the drugs are widely viewed as medicines for the relief of everyday tensions.

"Casual use of the drugs in this manner is medically unsound and constitutes a public danger. The tranquilizing drugs have not been in use long enough to determine the full range, duration, and medical significance of their side effects. Use of these drugs is no more to be encouraged than use of any other drug except where proper medical diagnosis determines that a drug is indicated to maintain the life and functioning of a person. **The prescribing of the drugs for emotional illnesses carries with it an obligation for continuing appraisal and supervision by physicians fully aware of the psychiatric symptoms involved and the potentials of their course of development, alteration or remission.**

"At the same time, it is important to be aware of subtle pressures that combine to foster public misunderstanding and misuse of the drugs. There is the eagerness of the public and of physicians themselves for 'good news' about a new treatment for psychiatric disorders. This tends to foster popular stories based on optimistic reports of early and limited research findings with the drugs, before such findings can be reproduced and validated by other colleagues. There is normal competition among pharmaceutical houses to refine, promote, and sell their own particular products. This competition should not involve physicians in public relations enterprises directed towards the premature announcement of 'successful use' of particular products.

"Persons engaged in any form of research or evaluation of therapy should be most dispassionate and objective in their pronouncements. It is suggested - - - - to be particularly alert to personal pressures (both internal and external) and circumspect in their announcements of early experimental results with the drugs."

Notes From Desk Of The Executive Secretary

Arizona AMEF Contributions

It is gratifying to learn that during the month of August $665.87 was contributed to the American Medical Education Foundation fund through the efforts of our Woman's Auxiliary. Pima County heads the list with $295.00; Maricopa followed with $117.00; Yavapai $90.00; and Yuma $79.87. The state auxiliary added another $84.00. Again the dependability of our fair ladies has been demonstrated. Congratulations and many thanks!!

AMEF — Advisory Member Appointment

The Board of Directors of the American Medical Education Foundation taking advantage of its by-laws provision has created a new membership in the Foundation.

"Advisory Members" are being chosen for their active interest in the Foundation and their prominence in the medical community. From this group will be selected a National Council which will aid and advise the Foundation on the best policies to pursue to assure its success. On recommendation to the Board of Directors, Doctor James D. Hamer of Phoenix, our Delegate to the AMA, is one of a first group elected to this membership. Congratulations — Doctor Hamer.

Hospital — Physician Relations

A.M.A. President Dwight H. Murray, speaking before the 58th annual convention of the American Hospital Association in Chicago during September had this to say: "I would be less than frank if I did not say that the medical profession is concerned by and vitally interested in the attempts by certain hospitals . . . direct or indirect . . . to inject themselves - - - in our opinion improperly - - - into the practice of medicine." Doctor Murray said that the hospital control of medical practice cannot possibly serve the best interests of the patients, but will only restrict or destroy the professional freedom and independence of the physician to practice the science and art of healing.

DOCTOR DRAFT

When the special doctor draft expires next July 1, physicians and other professional personnel covered by it still will be subject, up to age 35, to the regular draft. To discuss procedures to meet military needs after that date, the Defense Department has started conferences with representatives of groups involved, including American Medical Association, American Dental Association, the Association of American Medical Colleges and the four surgeons general — Army, Navy, Air Force and U. S. Public Health Service.

Although all physicians registered under the regular draft technically are liable for service up to age 35, few if any of those over 30 are apt to be called, as Defense Department estimates that the majority of its needs can be met by recent graduates of medical schools. One of the questions is whether any change in the basic Selective Service act will be necessary to facilitate the selective call-up of certain physicians and other specialists — including scientists — who are deferred for educational and occupational reasons.

THE TEN LEADING CAUSES OF DEATH — ARIZONA — 1955 INCLUDING DEATH RATES

Cause	Number of Deaths	Death Rate*
1. Diseases of Heart	2,168	222.1
2. Malignant Neoplasms, Including Neoplasms of Lymphatic and Hematopoietic Tissues	797	100.3
3. Vascular Lesions Affecting Central Nervous System	602	61.7
4. Certain Diseases of Early Infancy	483	49.5
5. Accidents – Other Than Motor Vehicle	342	35.0
6. Motor Vehicle Accidents	321	32.9
7. Influenza and Pneumonia	308	31.6
8. Tuberculosis	256	26.2
9. Congenital Malformations	155	15.9
10. Symptoms, Senility, and Ill-Defined Conditions	133	13.6
All Other Causes	1,771	
Total Arizona Deaths	7,518	770.3

*Deaths per 100,000 estimated population
Estimated Arizona civilian population 976,044 – July 1, 1955

Special report prepared for Arizona Medicine by R. M. Perkins, Bureau of Vital Statistics. 9-6-56.

Journal Report:

Hypertensive symptoms relieved in 96% of patients

"Comparison of pentolinium [ANSOLYSEN] with other preparations in 25 patients with severe essential hypertension, for whom all other methods of management had failed, showed that pentolinium is the most effective of available agents in reducing dangerously high blood pressure to the desired levels, and in modifying some of the complications of hypertension, as cardiac decompensation, cardiomegaly and retinopathy. . . .

"In 96 per cent (24 patients) clinical symptoms were relieved and the blood pressure maintained at comfortable levels. . . ."[1]

ANSOLYSEN®

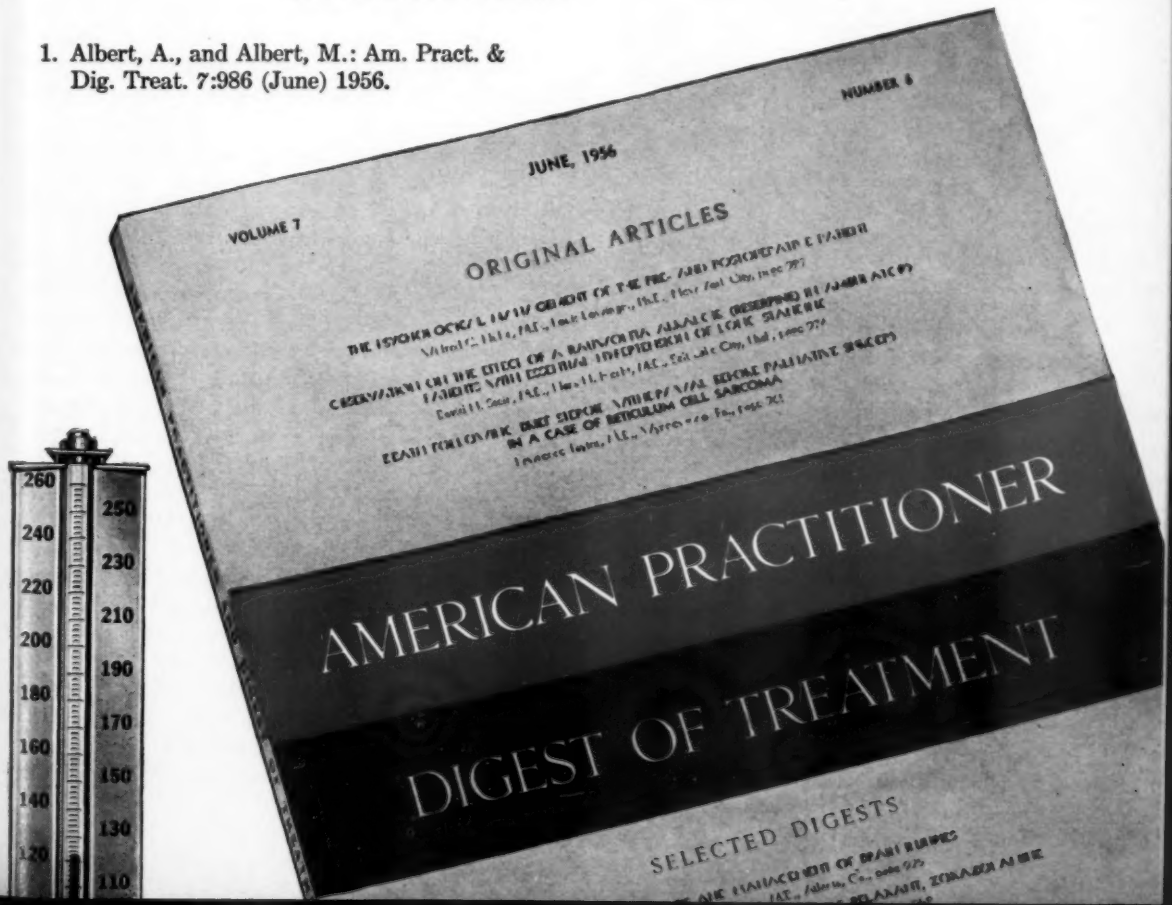
TARTRATE

Pentolinium Tartrate

Lowers Blood Pressure

Wyeth®
Philadelphia 1, Pa.

1. Albert, A., and Albert, M.: Am. Pract. & Dig. Treat. 7:986 (June) 1956.



1957 ANNUAL MEETING
Arizona State Medical Association
Stardust Hotel, Yuma, Arizona
April 10-13, 1957

INTRODUCING Leon Goldman, M.D., Chairman of the Department of Surgery, University of California School of Medicine in San Francisco, and participant in our 1957 Clinical Session.

Doctor Goldman is a native son of San Francisco, having been born there on February

Leon Goldman, M.D.

14, 1904. He received his Bachelor of Arts degree from the University of California in 1926, his Doctor of Medicine degree from the same institution in 1930, and his Master of Science degree in physiology from Northwestern University in 1939. He served his internship and residencies in surgery at the University of California and San Francisco hospitals from July 1, 1929 to June 30, 1933, and was a graduate student in physiology at Northwestern University Medical School from September 1938 to July 1939. Doctor Goldman has been affiliated with the University of California Medical School since July 1, 1933 when he became an instructor in surgery. He received his professorship in surgery in July 1950 and continues in that capacity to date. He has been Chairman of the Department of Surgery since July 1, 1956, together with Associate Dean of the School of Medicine.

Doctor Goldman serves on the staff of many San Francisco hospitals, is associated with a large number of medical societies and medical school committees. He became a Diplomate of the American Board of Surgery in 1940.

AMERICAN MEDICAL ASSOCIATION

A CORDIAL invitation is extended to all physicians in the West to attend the A.M.A. Clinical Session in Seattle November 27-30. An outstanding program has been arranged, and excellent hotel accommodations are available.

The center of activities will be Seattle's Civic Auditorium, where scientific sessions will be held and exhibits will be displayed. Conveniently located, the auditorium is easily accessible from downtown hotels, and transportation will be available for commuting to and from meetings. A cafeteria will be operated in the auditorium for midday meals.

Clinical Session headquarters will be the Olympic Hotel, where House of Delegates sessions and meetings of the Board of Trustees, Councils and Reference Committees will be held.

The scientific program will be beamed at the general practitioner, and subjects have been carefully chosen to be of interest and practical value. The program will include panel discussions, individual papers, motion pictures and closed-circuit television clinics.

Panel discussions will cover such subjects as hypertension, hemolytic anemia, prenatal care, problems of aging, epilepsy, low back pain, liver disease and vascular disorders. Twenty topics will be considered by panels of men prominent on the national scene and in the Northwest.

There will be 45 papers, considering fluid balance, urological problems, office psychiatry, varicose veins, fractures, diabetes, heart disease, and many other subjects. Contributors will include well-known medical educators and practicing physicians from all parts of the country.

The television clinics will include both wet (operative) and dry (non-operative) clinics. Talent will be drawn largely from Seattle because of the necessity of rehearsals and frequent briefing. There will be clinics on block anesthesia, treatment of burns, bleeding problems, intestinal obstruction, caesarean section, hand sur-

gery, hysterectomy, vein stripping and other subjects.

Scientific and technical exhibits have been arranged through A.M.A. headquarters. They promise to be as educational and interesting as in the past. Eighty-five scientific exhibits of the highest caliber will be on display.

Prepaid medical service plans of Washington, Oregon and Idaho will sponsor a hospitality suite in the headquarters hotel for three days during the session. An exhibit showing the prepaid plans' services to the public will be on display in the hospitality suite.

Plans have also been made for Auxiliary activities during the Clinical Session. Sight-seeing tours and other events are on the agenda.

Physicians are urged to make their reservations early. A reservation form will be found in the current issue of the Journal of the A.M.A. All reservations in Headquarters Hotel, The Olympic, must be made through A.M.A. headquarters.

1957 CANCER SEMINAR

Arizona Division, American Cancer Soc.
Phoenix, Arizona
January 10, 11, 12
Paradise Inn

Thursday Morning

Bone Tumors
David C. Dahlin, M.D.
J. Vernon Luck, M.D.
Eugene P. Pendergrass, M.D.

Mediastinal Tumors
Joseph Gale, M.D.
L. H. Garland, M.D.
Hans G. Schlumberger, M.D.

Thursday Afternoon

Ovarian Tumors
Dominic A. DeSanto, M.D.
L. H. Garland, M.D.
Joe Vincent Meigs, M.D.
Alfred Gelhorn, M.D.

Friday Morning

Relationship of Animal Tumors to Human Tumors
Hans G. Schlumberger, M.D.

Recent Advances in Chemotherapy of Malignant Diseases
Alfred Gelhorn, M.D.

Report of the President of American Cancer Society
David A. Wood, M.D.

Friday Afternoon

Clinical-Pathological Seminar
The Stomach
Joseph Gale, M.D.
David C. Dahlin, M.D.
Eugene Pendergrass, M.D.

Saturday Morning

Cytologic Diagnosis of Cancer of the Cervix
Joe Vincent Meigs, M.D.
Dominic A. DeSanto, M.D.
David C. Dahlin, M.D.
David A. Wood, M.D.

Use of Isotopes in Cancer Diagnosis and Therapy
John Z. Bowers, M.D.
L. H. Garland, M.D.

EDITOR'S NOTE: The agenda for the 1957 Cancer Seminar is outlined above, as announced by the Arizona Division of the American Cancer Society. This Seminar has increased in popularity each year and rightly so for the data presented is making this a clinical session well worth the attendance of the majority of the physicians here in the Southwest. We would like to strongly urge you to attend these excellent discussions.

SERVICE POLICY ARIZONA DIVISION — AMERICAN CANCER SOCIETY

DR. PAUL B. Jarrett, Chairman of the Service Committee of the American Cancer Society, Arizona Division, announced today that as part of its expanding program for Cancer Control, the Division has developed a new Service Program.

This program is to be put into action throughout the State as volunteer committees are organized. Its success depends on the volunteer help of many Doctors, the Medical Society, and the laymen. This help has already been pledged in most counties.

The purpose of the program is to provide professional and volunteer services to the indigent and medically indigent cancer patient, which will relieve unnecessary suffering and eliminate untimely death. This service to be provided as efficiently as possible through volunteer aid and to the extent to which funds are available after the needs of Research and Education have been met.

The Society's first step in putting the plan

into effect will be to survey the needs as indicated by the County Medical Society, Tumor Boards, Welfare Agencies, Hospitals, County Health Departments and the Voluntary and Governmental Agencies.

The Service Committee is now in the process of preparing a Manual, which will be a guide for the County Units as they activate this program.

Policies formulated by the Service Committee include the following:

1. American Cancer Society policy does not permit the payment of hospital bills, doctor's bills, x-ray or radium therapy, or nursing service while patient is in the hospital.

2. No funds of the Society will be used for an indigent patient unless all other possible sources of aid are not available for this patient.

3. In order to avoid duplication of existing community facilities the Society does not provide financial aid to patients eligible for County assistance; or to those eligible under any other program; or to patients who are financially able to meet their own obligations.

A. Whenever possible a patient should be referred to the proper community agency for assistance.

B. All indigent cases to receive financial aid be specially cleared with the local Welfare Department.

4. That no direct financial aid be given to a medically indigent patient. Payment can only be made to an Institution, Company or Agency.

5. The total expenditure for any one case shall not exceed $75.00 per year.

6. Financial aid can only be given for:

A. Medication (excluding experimental drugs).

B. Nursing — Visiting Nurse Service and Practical Nurses.

C. Dressings — free to all cancer patients, regardless of income, where requested by his physician.

D. Loan Closet equipment, hospital beds, wheel chairs and etc.

E. Transportation (for diagnosis and treatment).

F. Rehabilitation.

G. Referrals of patients to other community agencies for assistance.

H. Consultation for families with cancer problems (Information Centers).

7. No Case shall receive aid unless patient is under the care of a physician who is a member of the Arizona Medical Association.

8. Where adequate services are available within the State, no aid will be given for treatment or diagnosis out of the State of Arizona, except by prior approval of the Service and Executive Committees.

9. That in every County in the State, where adequate facilities exist, there should be a Tumor Consultation Service Committee, as defined by the College of Surgeons. That the Society shall offer its help to whatever extent it is available to local Medical Societies who wish to establish such a service.

10. That a Service Committee should be a standard part of each County Unit Organization, and that such a Committee should use the above rules as a guide for eligibility and care, since these rules have been recommended by the State Service Committee and approved by the State Board of Trustees.

The Board of Trustees of the Arizona Division, has defined a medically indigent patient as: "That individual who is unable to pay for his medical care and is not acceptable to the County or other Welfare Agencies for aid".

Because of the limitation of funds and the importance of meeting the financial requirements of the Research and education programs, this special asssistance program to the medically indigent must necessarily be an extremely limited one. It is estimated that there are 3,000 cases of cancer at any one time in Arizona. The first purpose of the Society is to save lives through a program of Research and Education. The Education program has first priority on funds to be expended in the local Unit program.

The new Service Program and Policy has been studied by the medical members of the Board of Trustees of the Arizona Division, American Cancer Society, and has met with their full approval.

Members of the State Service Committee are: Drs. Paul B. Jarrett, Melvin W. Phillips, Ian M. Chesser, Abe I. Podolsky, Zenas B. Noon, William E. Bishop, Warren Nelson, Guy Atonna, Douglas D. Gain, J. D. Hamer, and Mrs. Merle Farmer, Laura Dunshee, Margaret Maleady, Jean Reid, Grace Seaman, and Mr. Frank O. Williams.

SOCIAL SECURITY

IN RECENT months many physicians have heard from patients about the disability freeze provision in the social security law. This provision, added to the old-age and survivors insurance program in 1954, permits people who have prolonged total disability to apply to have their social security records frozen for the period of their disability. Thus, the time when they could not work and so had no earnings credited to their social security accounts does not count against them in determining their rights to benefits, nor the amount of benefits which will be payable to them at age 65, or to their families in case they should die.

Before a worker's social security record can be frozen, he has to meet certain work requirements. His social security record up to the time of his disability must show that he was in fact a worker, with a fairly regular and recent work history. In addition, he must be shown to have a medically determinable physical or mental impairment severe enough to keep him from engaging in any substantial gainful activity — one which has existed for more than 6 months, and is expected to last indefinitely or end in his death.

Securing The Medical Evidence Of Disability

The medical evidence needed to establish the nature and severity of the applicant's disability, the date it began, and its prognosis comes from the doctor who has treated the worker and knows his case, or the hospital or institution in which the worker has been confined. A Medical Report form was designed to assist the physician in furnishing the needed medical evidence and to indicate the nature and extent of clinical detail which would be necessary. It is given to the applicant for the "disability freeze" and he is asked to have it filled out by the physician most familiar with his impairment. The form itself is modeled closely after the medical report used by major life insurance companies in their disability claims work. In adapting it for use in the "freeze" program, the recommendations of a Medical Advisory Committee were closely followed. This Committee, composed of well qualified representatives of the medical and related non-medical professions, gives advice and guidance to the Social Security Administration on the medical aspects of the "disability freeze" program.

If you have received this medical form to fill out for any of your patients, you are probably aware that the law makes the disabled worker responsible for seeing that medical evidence is submitted for him and for paying any costs involved. The law does not permit the Government to pay any costs in connection with securing the medical evidence needed for a determination of disability. You may also know that to insure the confidentiality of the medical evidence, the medical report form is not to be returned to the patient, but is to be mailed by the physician direct to the local social security office. This office, incidentally, is ready to furnish additional information to the physician concerning the medical report form and the operation of the disability freeze.

Determining Disability

Determinations as to disability based on the evidence submitted are made under an agreement with the Federal Government, by professional mmbers of an agency of the State in which the applicant resides. In most States, this is the vocational rehabilitation agency. Since referral of disabled individuals for any rehabilitative services which might return them to gainful work is an important aspect of the program, each person applying for the social security disability freeze is told about the availability of vocational rehabilitation services.

On the professional team in the State agency at least one member is a doctor of medicine. The team reviews and evaluates all medical evidence assembled in the applicant's file, as well as such non-medical factors as age, education and occupational experience. Certain medical guides and standards, worked out with the advice of the Medical Advisory Committee are used in the consideration of the medical evidence. But, although these guides and standards can be applied in most cases, they are not rigid and arbitrary. The final determination in each case is based on all the available facts of the individual's impairment and vocational history, and, there is consultation among physicians in any borderline situation.

Guides to Filling Out the Medical Report Form

No matter how good the standards, nor how considered the judgment of the reviewing team, the determination reached can be no sounder than the evidence upon which it is based. To make sure that he is providing sufficient medical evidence for a prompt and fair determina-

tion, the doctor will want to consider the following guides in filling out medical report forms for those of his patients who have applied for the social security disability freeze:

First, include sufficient clinical detail to enable the reviewing team to make a sound determination as to the severity and extent of the patient's current condition;

Second, give enough of the clinical history to provide information as to when the disability began, and when it became so severe as to keep the patient from working;

Third, describe the probable course of the condition from now on, so that a decision can be reached as to whether the impairment is likely to continue indefinitely, or end in death, or whether it is self-limiting, or remediable in the forseeable future.

BOARD OF MEDICAL EXAMINERS — STATE OF ARIZONA

826 Security Building
Phoenix, Arizona

The Board of Medical Examiners of the State of Arizona at a regular meeting held July 21, 1956, issued certificates to practice medicine and surgery to the following doctors of medicine:

Alden, Neil Charles
1513 W. Thomas Rd., Phoenix, Arizona

Bassford, Paul Stanley, Jr.
Maricopa County Hospital, Phoenix, Arizona

Batts, Edward Lee, II
4139 N. 18th Drive, Phoenix, Arizona

Bill, Edward Charles
131 E. Barrymore, Stockton, California

Billings, Carl Emery, Jr.
2850 Santa Ynez Place, Tucson, Arizona

Brewer, Edward Allan
499 Pacific St., Monterey, California

Bromme, Dorothy Ann
532 N. Scottsdale Rd., Scottsdale, Arizona

Carstensen, Harold George
116 N. Tucson Boulevard, Tucson, Arizona

Class, Robert Nelson
57 N. Pennsylvania, Belleville, Illinois

Dean, Robert Theodore, Jr.
6007 North 16th Street, Phoenix, Arizona

Doyle, Peter James
2500 East Van Buren, Phoenix, Arizona

Eskelson, David Wright
240 La Mina Avenue, Ajo, Arizona

Evans, Robert Charles
St. Joseph's Hospital, Phoenix, Arizona

Fredell, Clarence Herbet
121 Aspen, Flagstaff, Arizona

Fulstow, Philip G.
P. O. Box 366, Kanab, Utah

Garcia, William
41 West Jackson, Tucson, Arizona

Gillette, John Murray
2440 E. 6th Street, Tucson, Arizona

Haeussler, William Bernard
3009 N. 19th Ave. — Apt. 124, Phoenix, Ariz.

Hazelhurst, George Nicholls
3818 E. Heatherbrae, Phoenix, Arizona

Helms, William Kendall
2104 Westward Boulevard, Phoenix, Arizona

Hodell, Frederick Howard
Maricopa County Hospital, Phoenix, Arizona

Hill, Richard Johnson
78 North 3rd Street, Buckeye, Arizona

Jensen, Thomas Walter
4013 North 6th Street, Phoenix, Arizona

Kahle, John Frank
505 North Beaver, Flagstaff, Arizona

Kimball, Howard Widtsoe
1602 West Roma, Phoenix, Arizona

Kudelko, Nicholas Michael
Box 66, Babbitt, Nevada

Lahr, Philip Heinrich
Maricopa County Hospital, Phoenix, Arizona

Lawler, Philip Wendell
2500 East Van Buren, Phoenix, Arizona

Leih, George Gustav Theodore
2125 West Hazelwood, Phoenix, Arizona

McCormick, Thomas Everett
321 West Central, Coolidge, Arizona

McCarey, Gladys L. Taylor
4320 East Thomas Road, Phoenix, Arizona

McMoran, Charles William
152 West Merrell Street, Phoenix, Arizona

Malone, Raymond Charles
29 B Street, Roswell, New Mexico

Munger, Arbor Day
1016 Sharp Bldg., Lincoln, Nebraska

Roberts, Loy Galatha
403 E. Glendale Avenue, Glendale, Arizona

Rydell, William Birger
40 W. Newton Street, Rice Lake, Wisconsin

Sandt, Karl Eugene
1750 Medical Arts Bldg., Minneapolis 2, Minn.

Spiritos, Michael Nicholas
615 Hillview Street, Winslow, Arizona

Stannard, Dale Hampton
608 Professional Building, Phoenix, Arizona

Stearns, Elliott Edmund, Jr.
2737 E. 21st Street, Tucson, Arizona

Taylor, Preston Johnson
1617 Wilson Ave., Salt Lake City, Utah

Walstad, Paul Marion
Route 4, Box 464, Turlock, California

Wrzesinski, John Thomas
800 North 1st Avenue, Phoenix, Arizona

ARIZONA BLUE SHIELD

TO ALL ARIZONA PHYSICIANS

TIME is drawing near for you to elect delegates to represent you at the Arizona State Medical Association meeting next spring. These delegates also constitute your official voice in running Blue Shield, since these same delegates along with the Professional Committee and Board of Directors serve as the Corporate Body of Blue Shield . . . and set its broad policies. Therefore voting for your state medical delegates is obviously of double importance to you.

Please note, however, that the House of Delegates is not your only means of operating Blue Shield. You have an elected all-Physician Professional Committee representing all fields of practice and all parts of the state. These men *work* long and hard for you and entirely handle the medical affairs of Blue Shield such as fee schedules and adjudication matters. They are paid only in the satisfaction that they are doing a service for the people of Arizona and you as well. Please advise them of your wishes. Also you are free to write or contact personally any or all of the predominantly-physician Board of Blue Shield, its officers, or the recently appointed Joint Commission for the study of the Arizona plan (Chairman, Dr. Hilary Ketcherside, Phoenix). An able and dedicated administrative staff stands ready to carry your wishes into actuality. Being informed is important. Your desires or requests for information are as close to you as pen, ink, and paper, or the telephone.

But to get back to the delegates. Please consider how important this delegate is to you. We have almost always had a good House of Delegates, so let's keep it that way or go a step further and have even better representation. A good delegate doesn't need to be the winner of a personality contest. He may be, but not necessarily, the fellow who tells the best stories in the hospital locker rooms. He may be a specialist or a general practice man, both have minds with which to think. He is often an older man experienced in running the affairs of the local County Society; but he need not be. Young men with new ideas are desirable to temper the conservatism of the old guard. The man you select should be:

Interested
Fair-minded
Able to think clearly
Able to stand on his two feet and deliver his thoughts,
Sincere, dedicated
An improver, not a wrecker;

and above all he must be there to tell your story and to run things right. To quite this election year's most potent slogan — "Vote for the right man."

WHAT IS HAPPENING AROUND BLUE SHIELD THIS MONTH?

1. The new building being constructed on Indian School Road near 3rd Avenue in Phoenix is well on the way. We have branch offices in Tucson and Flagstaff now.

2. As an outgrowth of deliberations of the Joint Commission, it is likely that a research organization such as Stanford Research Institute will be employed to make a study of the future scope and intent of Blue Shield.

3. Four different physician groups have prepared, checked and double checked and completed a list of procedures whereby assistant surgeons will be paid a fee, but not at the expense of the surgeon's fee.

4. The Professional Committee, after long, careful deliberations, has just completed an entirely new fee schedule which, for the most part, provides many increases and eliminates previous inequities.

5. The work of other committees to broaden Blue Shield coverage is well along the way to completion. If the laggards involve you, get after them. A prod is far more productive than a gripe.

6. New contracts embodying the above changes and to meet previous objections are being drafted by the staff and legal counsel and should be ready to replace the old contracts some time after the first of the year.

It takes time but progress is being made . . . as rapidly as possible. Other things are in the offing, things that we are sure you will approve. So "buttonhole" your delegate, write us letters, come in personally; understand Blue Shield, and make it just as you want it.

G. Robert Barfoot, M.D.
Pres. Ariz. Blue Shield

THE PHYSICIANS FORUM

THE following paragraphs are reprinted from the Secretary's Letter of the American Medical

Association. They deserve your consideration.

"The Physicians Forum, which was once spearheaded by a committee headed by the late Ernst P. Boas of New York, is passing the hat again among doctors for funds to finance a campaign to get social security for doctors. The Forum recently mailed a four-page brochure to every doctor in the United States, practically the same kind of brochure that it mailed back in 1952.

"The response from doctors will probably be no better this time than it was in 1952. Too many honest and sincere physicians recall the activities of The Physicians Forum a few years back.

"The Forum, which labeled itself the "voice of the liberal doctor," once championed the fight in behalf of the Wagner-Murray-Dingell bill providing for compulsory national health insurance. Many physicians also remember how, in November, 1945, The Physicians Forum issued a statement saying that it 'strongly approves the message of President Harry S. Truman calling for the establishment of a nationwide health and medical care program to supply the medical needs of all Americans regardless of income, race and religion.'

"Within the recent weeks, the A.M.A. Journal published a series of two excellent articles dealing with the subject of social security for physicians. The articles strongly refute the statements and arguments presented in The Physicians Forum brochure, showing beyond question that they are false and misleading."

THE ARIZONA MEDICAL ASSOCIATION, INC.

826 Security Building
Phoenix, Arizona

Location Opportunities

AJO — Pop. 7500 — General-industrial practice. Has need of general practioner with special interest in internal medicine, to complete four man staff, with 47 bed modern well-equipped industrial hospital. Salary and income guarantee. Write Dr. F. A. Nelson, Chief Surgeon, for particulars.

ASHFORK — Pop. 700 — Railroad center — Contact Mr. J. J. Slamon, Justice of Peace, Ashfork, Arizona.

DOUGLAS — Pop. 10,000 — Opportunity for associate practice in OALR. Contact James S. Walsh, M.D., 631 9th Street, Douglas, Arizona.

FLAGSTAFF — Pop. 7500 — Navajo Ordnance Depot is in the process of recruiting for a medical officer GS-12, $7040 per annum. For further information write M. R. Bell, Personnel Officer, Navajo Ordnance Depot. Flagstaff, Arizona.

FLAGSTAFF — Excellent opportunity for a pediatrician and for a radiologist. Please contact Morris M. Zack, M.D., 411 Birch Street, Flagstaff, for further information.

GILA BEND — Pop. 2500 — Good opportunity for general practitioner. Cattle, cotton, and general farming. Office and equipment available. $150 monthly income from Board of Supervisors. Contact Mrs. J. F. Allison, Box 126, Gila Bend, Arizona.

KINGMAN — Pop. 3342 — Excellent opportunity for a General Practitioner-Surgeon to take over the active practice of a doctor leaving there. Equipment available at nominal fee. If interested, contact Doctor Walter Brazie, Masonic Building, Kingman.

SAN MANUEL — Physician to be associated with the copper mining company located there. Contact Francis M. Findlay, M.D., San Manuel Hospital, San Manuel.

TUCSON — An opening in the Outpatient Department of the Veterans Administration Hospital in Tucson for a generalist or internist will occur early in September. State License is necessary, but not necessarily an Arizona license. If interested contact S. Metzer, M.D., Director, Professional Service, V.A. Hospital, Tucson, Arizona.

TUCSON — Opening for a Board certified or Board eligible Orthopedist to form and head an Orthopedic Department in the Tucson Clinic. If interested, contact D. J. Heim, M.D., The Tucson Clinic, 116 North Tucson Blvd., Tucson, Arizona.

TUCSON — Looking for a General Practitioner for plant services — $750.00 monthly, 5 days a week. Contact Doctor Meade Clyne, 116 North Tucson Boulevard, Tucson, Arizona.

WILLCOX — Pop. 900 — Doctor C. J. Bozzi, 124 West Maley Street, Willcox, Arizona, is interested in disposing of practice. He is expecting only reasonable payment for equipment. Records go with such sale.

WILLCOX — In need of a medical doctor badly. Contact Mrs. John C. Wilson, Box 548, Willcox, Arizona.

YOUNGTOWN — Pop. 130 — Located 16 miles from Phoenix, 4 miles from Peoria, 1½ miles from El Mirage, 1 mile from Surprise, each a potential field of practice. It is within an agricultural area. Most residents are 60 years of age or older, and are in need of medical care. Currently provided at no rental is office space. A medical center facility is being planned. Interested medical doctors may contact Mr. Sid Lambert, Box 61, Marionette, Arizona.

YUMA — Pop. 15,000 (approximately) — In need of a county physician. This is an ideal set-up for a retired or semi-retired doctor. The doctor could devote all his time to the job or have a private practice in addition. If interested, call Mr. Robert Odom collect at SUnset 3-7843 as soon as possible.

For Information On Opportunities In the Feld Of

Industrial Medicine, Contact:
F. R. Nelson, M.D., Phelps Dodge Hospital, Ajo, Arizona
Carl H. Gans, M.D., Phelps Dodge Hospital, Morenci, Arizona
Ira E. Harris, M.D., Miami-Inspiration Hospital, Miami, Arizona
Charles B. Huestis, M.D., Box 928, Hayden, Arizona
Elvie B. Jolley, M.D., Copper Queen Hospital, Bisbee, Arizona
H. W. Finke, M.D., Magma Copper Company Hospital, Superior, Arizona
Francis M. Findlay, M.D., San Manuel Hospital, San Manuel, Arizona
John Edmonds, M.D., Kennecott Copper Corporation Hospital, Ray, Arizona

County - State - National Medical Society Meeting Calendar

APACHE — 2nd Tuesday Monthly (3-Way Telephone Communication) Special Meetings at McNary.
COCHISE — 3rd Wednesday Monthly (Alternating Between Bisbee-Douglas Commencing September)
COCONINO — 2nd Tuesday of March, June, September and December (8 P.M. Flagstaff Hospital)
GILA — 3rd Thursday Monthly (excepting Summer Months) at 7:00 P.M.
GRAHAM — 1st Monday Monthly at 8:00 P.M. — Special Meetings Subject to Call.
GREENLEE — 3rd Tuesday Monthly Evenings — Special Meetings Subject to Call.
MARICOPA — 1st Monday Monthly Evenings (September through June) Good Samaritan Hospital Auditorium.
MOHAVE — Subject to Call.
NAVAJO — Monthly Subject to Call at Winslow Memorial Hospital or Nancy Wright Clinic.
PIMA — 2nd Tuesday Monthly Evenings (October through May) El Conquistador (excepting December at Davis-Monthan AFB and May at VA Hospital).
PINAL — 2nd Tuesday Monthly (September through June) at Pinal General Hospital, Florence.
SANTA CRUZ — 3rd Thursday Monthly Evenings (September through May).
YAVAPAI — 1st Tuesday Monthly Evenings (September through May).
YUMA — 1st Tuesday Monthly Evenings (September through June).
ARIZONA MEDICAL ASSOCIATION — 66th Annual Meeting — Stardust Hotel, Yuma — April 10 through 13, 1957.
AMERICAN MEDICAL ASSOCIATION — Clinical Meeting — Seattle, Washington — November 27 through 30, 1956.
Annual Meeting — New York, N. Y. June 3 through 7, 1957.

HILL-BURTON GRANTS

The Department of Health, Education and Welfare reports as of July 31, 1956, the status of all Hill-Burton grants for the State of Arizona. Approved, but not yet under construction are fifteen (15) projects at a total cost of $12,050,437, including federal contribution of $3,650,050, designed to supply 884 additional beds. Eight (8) projects are under construction representing a total cost of $2,844,201 (federal contribution of $1,003,085 included), designed to supply 30 additional beds.

Marking the 10th anniversary of enactment of the Hill-Burton Act, officials of the Department of Health, Education, and Welfare estimate that during that period a total of 3,047 projects have been approved. Their cost is placed at $2,467,333,207, with the federal share at $781,421,267. Breaking this total down, hospital projects under the regular Hill-Burton program amount to 2,843 at a cost of $2,342,545,709; the federal share was $752,938,357 or roughly one-third of the total. Medical facilities authorized under the 1954 amendments accounted for 204 units at a cost of $124,787,498; the federal share this time was $28,482,910 or about one-fifth of their cost. Medical units are divided: 42 chronic disease hospitals, 42 nursing homes, 77 diagnostic centers and 43 rehabilitation centers.

Looking ahead, the agency insists the "bed deficit of the nation is still great" and that estimates by states reflected in state plans indicated about 843,000 more hospital beds are still needed.

U.S.P.H.S. — Arizona Contract Care Payments

Below is tabulated a summary of government funds expended by the U. S. Public Health Service to physicians, hospitals and other vendors in Arizona during the fiscal year 1956. "Other Vendors" include laboratory services, orthopaedic appliances, eye glasses, etc.

	Physicians and Surgeons		Hospitals and Institutions		Other Vendors	
County	No.	Amount	No.	Amount	No.	Amount
Arizona - Total	85	$33,342.23	19	$116,127.33	40	$10,624.47
Apache	—	——	2	345.00	—	——
Coconino	—	——	1	4,979.30	1	24.40
Gila	5	936.18	1	386.25	1	48.75
Graham	3	700.00	1	1,106.15	—	——
Maricopa	35	7,872.35	7	86,362.79	21	4,704.87
Mohave	2	6,229.50	1	6,128.25	2	507.00
Pima	26	10,272.70	4	7,686.75	6	1,857.50
Yavapai	6	4,041.00	1	6,267.84	2	233.00
Yuma	7	3,290.50	1	2,865.00	7	3,248.95

Woman's AUXILIARY

CIVIL DEFENSE — WHAT TO DO IN CASE OF AN ENEMY ATTACK

IF YOU LIVE BEYOND THE SAFETY LINE

REMEMBER, no one can be sure just where an enemy bomber may strike. If your home is near the Safety Line on the map, it is better to move still farther out in an evacuation. A good storm cellar 20 miles outside the city will give you and your family protection. If in doubt, MOVE OUT when advised to do so.

MAKE A PLAN

Your plan will help you most in such an emergency. You should have worked it out with your family so that each one knows what to do, where to go, and what to take along. A civil defense survival kit would be a good thing to have ready. Make sure that your plan conforms to the community plan. Try it out some day to see if you have forgotten anything important.

WHAT TO DO

When evacuation is advised, close your home as you would if you were going to be away for several days. Take the things you have assembled for such an emergency to your car and drive on the nearest evacuation route. Do not speed, drive carefully, and when you have entered the evacuation route, drive along at the same speed as other traffic. REMEMBER, it will do no good to try to get ahead.

EVERY CAR MUST BE FULL

If you can't find a ride start walking along an escape route. Organized public transportation may be able to pick you up, or you may be able to obtain a ride with others. Drivers pick up passengers to capacity. Drivers of unassigned trucks and busses swing by nearest school or hospital and load to capacity. Get as far away as possible, beyond the 15 mile line for sure. Then find the best shelter.

TRAFFIC RULES . . . MOVE OUT . . . NEVER ACROSS

When the "YELLOW ALERT" signal sounds and you decide to evacuate, all escapeways will be one way — outbound only. Do not try to cross any of these escapeways or police will stop you. For another reason, traffic going outbound would be so heavy you couldn't cross if you tried.

Head in one direction only — outbound, the way traffic is moving. Disregard any fixed signals that would prevent you going outward.

Keep your car radio on 640 or 1240 at all times. This is your only communication.

Keep ½ tank of gas at all times

Have in the car the following:

1. DRINKING WATER
2. FOOD
3. FIRST AID SUPPLIES
   BLANKETS, READING MATERIAL
   DESERT SURVIVAL PAMPHLET
   EVACUATION MAP
   RADIO

SECURE EVACUATION MAPS AND OTHER INFORMATION AT FIRE STATIONS, BANKS, FRATERNAL AND VETERANS ORGANIZATIONS AND ANY C. D. HEADQUARTERS

ONCE OVER QUICKLY

1. The Yellow ALERT SIGNAL means you should tune your radio to either 640 or 1240 on the dial to receive Civil Defense Instruction. You should also start moving out of town. The YELLOW ALERT SIGNAL will be announced by a 5-minute steady sound on the air raid warning sirens, and also by the radio (640 or 1240 on your radio dial).

2. Package now the essential items you wish to take with you.

3. Ride out if you can. Walk out if you have to.

4. Prearrange evacuation transportation for your family (at home, at work, at school, etc.).

5. Study the map and familiarize yourself and your family under various conditions, (at work, at home, etc.) keeping in mind the district in which each person might be at the time. Select your family's rendezvous.

6. Get on escapeways promptly — but calmly. Pick up passengers to capacity.

7. Follow traffic — one way only, outbound!

8. Don't try to cross escapeways.

9. Don't use the telephone.

10. Take cover on WARNING RED SIGNAL (a three-minute wailing sound that rises and falls in intensity).

11. The ALL-CLEAR SIGNAL is no longer in use because of possible lingering radiation in some areas. You will be notified by radio or by CIVIL DEFENSE BLOCK WARDENS when it is safe to come out of your shelter or return to city.

YOU WILL WANT TO KNOW ABOUT AIR RAID WARNING SIGNALS

All citizens should familiarize themselves with the two types of warning signals which will be sounded by CIVIL DEFENSE OFFICIALS in times of emergency. They are: The Alert — a steady blast of 5 minutes duration. The alert signal will indicate to the public to act according to the operations plan of the local CIVIL DEFENSE OFFICE.

1. YELLOW — 5 minutes — evacuate city
2. RED — 3 minutes — take cover

YOU WILL WANT TO KNOW ABOUT CONELRAD

Where will you get immediate and official information from CIVIL DEFENSE authorities in times of emergency?

You will get this vital information BY TURNING THE DIAL OF YOUR RADIO TO 640 or 1240 ON THE DIAL. Remember these numbers, they may save your life.

Because of the H-bomb, best measures for safety consist of getting out. The farther out you are the better your chances of survival.

(Editor's Note — The above recommendations are sound. They are of particular significance to the people of Phoenix and Tucson. But — Tucson and Pima County are laggard. The people of the Tucson area have no chance to evacuate, they have no alarm system. Evacuation is a must, it is feasible. But — they have not budgeted $0.07 per person to give themselves this chance!)

ARIZONA CIVIL AIR PATROL

1. Physicians are needed in the Arizona Civil Air Patrol as Medical Officers.

2. CAP, as Auxiliary to the USAF, has the following missions:

A. The Training of Air Cadets; Summer Encampment; Foreign Exchange.

B. The Perforance of Disaster Rescues.

C. Cooperation with Civil Defense.

3. CAP is a Volunteer Organization whose members serve without pay. Reserve Officers are entitled to their Reserve Rank; others are given appropriate rank.

4. CAP began in 1941 in response to the need to help patrol our coastlines to spot enemy submarines.

5. CAP organization is based on a Natl. HQ at Bolling Field, Washington, D. C.; with Wing HQ in each State and Territory; and with Groups and Squadrons within each State.

6. There are several Groups and Squadrons in Arizona, as; — Phoenix, Tucson, Prescott, Casa Grande, Yuma, Wilcox, Eloy, Kingman, Ft. Defiance, Window Rock, Winslow, Kayenta, Bagdad, Bullhead City, Morenci, Florence, Mesa, Holbrook, Payson, and Buckeye.

7. Qualifications for CAP Medical Officers; CAPR 160-1 dated 31 Jan. 1955. National HQ CAP AUX of USAF Bolling Air Force Base Washington, D. C.

8. Any physician who is interested in joining CAP may apply to his nearest CAP Unit Co. J. H. Stickler, M.D., Lt. Col, Wing Medical Officer, Tucson Municipal Airport.